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Listerian Oration.[1]

LISTER: SOME ASPECTS OF HIS LIFE, WORK AND CHARACTER.

By SIR HENRY NEWLAND, C.B.E., D.S.O., F.R.C.S.,
Adelaide.

Stands genius a deathless adornment,
A name not to be worn out with the years.

I MUST in the first place thank the Council of this Branch for the great honour they have done me by inviting me to deliver the Listerian Oration. It is approximately twenty-one years since I, when Secretary of this Branch, suggested to the Council the form the memorial to Lord Lister should take. I then little thought that I should ever be deemed worthy of the honour of delivering the oration on the occasion of the attainment of its majority.

This circumstance, it seems to me, makes it fitting that this address should deal with some aspects of Lister's life and work rather than with surgical experiences of my own.

To most men Lister is becoming a vague and shadowy figure. But the truths of antiseptic surgery are eternal, and it is hard to conceive that the memory of the master surgeon who first demonstrated them will ever fade. Sir Thomas Browne recognizes the ephemeral nature of fame in the following passage in "Urn Burial":

> But the iniquity of oblivion blindly scattereth her poppies, and deals with the memory of men without distinction to merit of perpetuity . . . . Oblivion is not to be hired. The greater part must be content to be as though they had not been, to be found in the register of God, not in the record of man.

[1] Delivered at a meeting of the South Australian Branch of the British Medical Association on May 31, 1934. (Received for publication, February 5, 1935.)

Lister's memory is surely immune to oblivion's opiate, for the aseptic healing of all wounds will be man's object as long as surgery lasts, and so long shall men do him homage.

It may be that I am the only person in this room who has ever seen the subject of this address. I trust that I shall not try your patience by recounting the three occasions on which I had that good fortune.

The first time was in 1899, when I was living with the late Mr. Jonathan Hutchinson, junior, at Number 1, Park Crescent, Regent's Park. Lord Lister lived at Number 12, and one day his distinguished figure was pointed out to me as he was entering his carriage for a drive in the park.

I next saw him at the centenary dinner of the Royal College of Surgeons of England, of which Osler has given an interesting account:

> At eight a dinner was given by the College in the hall of Lincoln's Inn. It was the best ordered dinner I have ever attended. We sat down about 8.15 and rose about 11.15. A more distinguished company has perhaps never been gathered to do honour to the profession. To the right of Sir William sat the Prince of Wales, the Marquis of Salisbury, the Duke of Northumberland, Lord Strathcona, Lord Kelvin, and a group of Honorary Fellows. To his left sat the Duke of Cambridge, Earl Rosebery, the Lord Chancellor, Lord Lister, the Lord Mayor and other Honorary Fellows. The members of the Council occupied seats at the ends of eight long tables. Among many excellent features of a most exceptional dinner may be mentioned the shortness of the speeches and the softness of the music. To Lord Lister, with his Quaker upbringing, one speech would especially appeal. Lord Rosebery, witnessing the harmony existing in the medical profession throughout the world, expressed the hope that perhaps through science might be realized that peace on earth to effect which all other means had failed.

The last occasion on which I saw Lister was also a great one. The second International Congress on Tuberculosis in London was in progress. An outstanding and somewhat disconcerting episode occurred on the second day, when Koch, after being introduced by Lord Lister, made his famous statement that human tuberculosis was practically not transmissible to animals; that the reverse was probably also true, and that therefore the attempt to stamp out the disease in cattle as a source of human infection had been misdirected. In the discussion that immediately followed the address, Lister, with extreme clearness of thought, promptly put his finger on the weak point in the deduction Koch had drawn from his experiments.

Surgery before Lister.

"Prior to Lister, the history of suppuration is the history of surgery" remarks the writer of a fascinating article "On the Healing of a Wound", in a recent number of *The Quarterly Review*. Most of what I am immediately about to say is culled from that article.

> The mediæval surgeon strove for suppuration; and the beneficence of suppuration arose from the doctrine of the four humours. Pus was considered to be a crudity or coction of those imaginary qualities. Indeed it still goes by the name of humour, and the French speak of a suppurating scrofula as *l'humeur blanche*. The formation of pus was encouraged to relieve the system of an inherent crudity. "To get it out of the system" we still say. Poultices, grease, and salves of saintly potency with names such as St. Paul, St. Thomas and St. Victor were applied to wounds, Galen brought the balm of Gilead from Damascus, and Friar's Balsam, once known as Crusader's Salve, is yet used by us.

There is a beautiful piece of sculpture by an ancient Greek master which has reference to suppuration. It represents the famous archer Philoctetes endeavouring to find relief by fanning with a bird's wing his festering foot, which led to his being left at Lemnos when the Greeks sailed for Troy.

> The great surgeons who tower above the base crowd of bunglers strove for primary healing, but for centuries they were borne down by the false readers of Galen who followed the method of suppuration as nature's only cure. Roger of Salerno was their leader; he promoted coction by suppuration and dressed wounds with galenical salves. Surgery took the wrong turning and remained in the wrong course until our own time.

Powerful voices had been raised in protest many centuries before. Celsus in the first century warns the surgeon, in his anxiety to exclude the air, not to use the suture until the depth of the wound was so cleaned that no clot remained, for this formed pus, excited inflammation and so prevented union. The most powerful voice was that of Theodoric in 1266: *"qui pulcherrimas cicatrices sine unguento aliquo ducebat"*. "It is not necessary", he insisted, "that pus should be generated in wounds; no error could be greater; it hinders nature, prolongs the disease, and prevents the healing of the wound." Theodoric was a true precursor of Lister. Henry of Mandeville was his successor, with the dogma: "Wash the wound free from all foreign material; use no probes or tents; apply no oily or irritant substances; avoid the formation of pus, which is not a process of healing." "Many more surgeons", he said, "know how to cause suppuration than to heal a wound. Avoid everything likely to cause pus, *'medicinæ quæ faciunt nasci pus'*." He and his followers washed the wound with wine; they removed every foreign body; they brought the edges together and did not allow even the wine to remain within. "Nature", they said, "supplies the means of union in a viscous exudation"—"natural balm", as Paré called it. Fibrinous exudation we would call it today.

A belief in the virtues of suppuration, notwithstanding voices raised in opposition, prevailed down to our own time; surgeons strove for a pus which they called laudable. Sir Thomas Clifford Allbut in 1904 stated:

> In the third quarter of the nineteenth century, in my days as a physician, the apothecary of a large hospital showed me a row of amputations with stumps pouring out pus in cataracts upon the cushions, and exclaimed: "That, sir, is what I like to see; nothing so wholesome in a wound as a good discharge of laudable pus."

I suppose it is not far from the truth today to say that the average medical man's knowledge of Lister may be summed up as follows. He knows that Lister was the inventor of the antiseptic method of treating wounds and that the method is based on the "germ theory". He knows, too, of

Pasteur's researches. He perhaps knows that Lister's attention was first directed to those researches in the course of a walk with a professor of chemistry and that Lister chose carbolic acid because it had been successful in treating the sewerage of Carlisle.

It is not at all well known how superb was Lister's intellectual endowment and how great were his attainments as a trained observer and experimental physiologist long before he came to devote himself to the particular work which made him famous. It is to this earlier phase of his work that I especially wish to draw your attention, but before I do so it is essential, if Lister's personality is to be understood, to refer to still earlier biographical details.

The First Phase.

His parents belonged to the Society of Friends. His mother came of sturdy north country stock and before her marriage had taught in a Quaker school. His father was a Yorkshireman who had succeeded in business as a wine merchant. By virtue of their faith unable to join the army or navy, and forbidden by the law of the land to enter the universities, the ablest of the Quakers had for generations past perforce employed their talents in trade. Owing to their integrity and natural frugality they had become a Quaker aristocracy of wealthy bankers and brewers. Lister consequently never suffered from any financial embarrassment.

Even as a child Lister was bent on becoming a surgeon, and quite early showed his aptitude for the profession by dissecting and drawing skeletons. One of his efforts was to articulate the skeleton of a frog, which, he said, "looks as if it were going to take a leap". It will appear later on how deft he was in the use of his pencil.

At seventeen he went to University College and entered for the degree in arts as a preliminary to that in medicine, a sequence which, though time-consuming, has, even in these fleeting days, much to recommend it. University College differed from Oxford and Cambridge in that it was founded for the purpose of giving an unsectarian education free from the ecclesiastical influences which pervade those universities, and by implication it was to be the home of a liberal theology, tinged even with rationalism, if the opinion of its leading spirits indicated anything.

It was doubtless the college's association with a hospital that attracted Lister. The great Liston was still at its head, and in 1846 Lister saw him perform his first operation under an anæsthetic. The hard, incessant work of four years led to a breakdown in Lister's health and spirit after taking his B.A. degree. A severe attack of smallpox and a wave of introspection made matters worse. His father treated him with rare insight. He sent him to Ireland with the advice that "thy proper part now is to cherish a pious, cheerful spirit, open to see and to enjoy the bounties and the beauties spread around us . . . not give way to turning thy thoughts upon thyself, nor even at present dwell long on serious things". Lister began his medical course in 1848 amid congenial surroundings. Warned by his breakdown, for the rest of his life he took regular holidays at home or abroad. In 1851 he spent a month at Shanklin, in the Isle of Wight, in bathing and in shrimping for microscopic purposes. He was evidently no mean swimmer, for when he became a dresser in the surgical wards at he tells that he swam fifty-two yards in two minutes.

Lister's first introduction to septic diseases was University College Hospital. Watson Cheyne states that his very first patient had been attacked by phagadenic gangrene, one of the most terrible of septic diseases. That and other similar cases impressed him so greatly that he never forgot them, and always felt that no matter was more urgent or might be more fruitful than the attempt to find out the cause, the prevention and the best methods of treatment of those septic diseases. It must, I think, have been in search for the cause that, some years later, he began right at the beginning, as it were, and studied the coagulation of the blood and the early changes in inflammation.

According to the times, University College Hospital was up to date in 1851, when Lister was getting his first experiences of operative surgery, but the equipment was actually very primitive. The operations for the week were done on a Wednesday afternoon in a small room with a single gas jet, one basin and a wooden table.

At the age of twenty-five Lister had taken his degree in medicine and had become a Fellow of the Royal College of Surgeons of England. He had already proved his skill as a microscopic anatomist by his confirmation of Kolliker's discovery that the iris was an involuntary muscle. In it he showed the presence of separate dilator and sphincter fibres. That he was no mean draughtsman his illustrations of these fibres and of the tiny muscles that act on the hairs of the scalp well show.

The Second Phase.

In September, 1853, Lister decided to spend a month or two in Edinburgh and took a letter of introduction, no doubt couched in very favourable terms, from Sharpey to his friend Syme. The latter, then aged fifty-four, was in the zenith of his career and was generally considered the first surgeon in Europe. The impression Lister made on Syme was a good one, for he was forthwith invited to dinner and was soon quite at home in the Syme *ménage*. Lister was then twenty-six, and he speedily became a great admirer of Syme and found grace in his sight, as Joseph of old had done with Potiphar. Being a goodly person and well favoured, Joseph the Quaker found grace as well in the sight of Syme's daughter, Agnes. As a man of peace he hated, however, the controversies in which Syme delighted. "In fact, I doubt if I could do it", he wrote, "though I have never tried much." Lister, when roused, would warmly defend what he was convinced was the truth, but he was never auto-

cratic or made excessive claims. He was modest, but well able to uphold his views. He was never conscious of having been endowed with the eye of genius, and when he was twenty-eight he confided to his father: "As to brilliant talent, I know I do not possess it, but I must make up as far as I can by perseverance." Lister had unquestionably a great capacity for work.

> For sluggards' brow the laurel never grows,
> Renown is not the child of indolent repose.

The Edinburgh infirmary, with three hundred beds (more than three times those of University College), and the opportunities Syme gave him there as well as in private afforded Lister occupation and happiness. Thus he writes to his father: "Thou canst hardly conceive what a high degree of enjoyment I am from day to day experiencing in this bloody and butcherly department of the healing art." At Christmas the absence of Syme's house surgeon afforded Lister the chance of filling the position, a humble one for a Fellow of the Royal College of Surgeons in England. Syme, however, treated him as a colleague and gave him full discretion to operate on all emergency cases at night. After taking the Edinburgh fellowship and attending a course of operative surgery in Paris, Lister became betrothed to Agnes. This led him to leave the Quakers and become a member of the Episcopalian Church of Scotland. His father, who we have already seen was a master of tact, wrote to him: "I trust we shall be very careful to say nothing in disparagement of those whom we shall probably find on acquaintance to be our superiors." At this time, nearly ten years before he was to bring his method of antiseptic treatment of wounds to the notice of the profession, he was engaged in trying to discover what happened in the early stages of inflammation. I emphasize this fact because it shows how long and earnestly he devoted his attention to the causes of inflammation and its pathological phenomena. The leg of the frog lending itself to the study *in vivo* of his purpose, he spent "glorious nights" watching the changes in the tiny blood vessels in health and after he had applied various irritants to the web. The changes he observed were described in a beautifully illustrated paper read before the Royal Society. The facts he stated are as true today as when he described them. The paper is a surgical classic and teems with experiments. No man ever obeyed more faithfully the injunction of Harvey to "study and seek out the secrets of nature by way of experiment", or conformed to Harvey's motto for a young man: "*Dii laboribus omnia vendunt.*" His work on the coagulation of the blood is another example of his *flair* for experimental physiology. Lord Moynihan, with feigned self-pity, has said that he himself is "a physician condemned to practise surgery". We may be thankful for the good of mankind that Lister was a distinguished physiologist condemned to the same fate. I had written these words when I came across Sir Michael Foster's opinion of Lister: "One whose career as a practical surgeon we physiologists selfishly deplore, for we see in him a puissant captain."

Those of you who remember your pathology will see that the following account by Lister of the early stages of inflammation pretty closely coincides with your recollection.

> The effects produced upon the circulation by the application of an irritant to a vascular part are twofold, consequent upon two primary changes in the tissues, which, though often concomitant, are entirely independent both in nature and mode of production. One of these is dilation of the arteries, commonly preceded by a brief period of contraction, giving rise, in proportion to the increase of calibre, to more free flow through the capillaries, the blood remaining unaffected, except in its rate of progress. The purely functional phenomenon is developed indirectly through the medium of the nervous system, being not limited to the part acted on by the irritant, but implicating a surrounding area of greater or less extent. The other change is the result of the direct operation of the irritant upon the tissues, which experience some alteration in consequence of which the blood in their vicinity becomes impaired. It loses the properties which characterise it while within a healthy part and which render it fit for transmission through the vessels, and assumes those which it exhibits when removed from the body and placed in contact with ordinary solid matter. The first indication of this disorder of the vital fluid is that its corpuscles, both red and white, acquire some degree of adhesiveness, which makes them prone to stick to one another and to the vascular parietes and, lagging behind the *liquor sanguinis*, to accumulate in abnormal numbers in the minute vessels. This adhesiveness may exist, in proportion to the severity of the affection, in any degree; from that which merely gives rise to a very slight preponderance of the corpuscular elements of the blood in the part, up to that which induces complete obstruction of the capillaries. When the irritation has been very severe, the *liquor sanguinis* also shows signs of participation in the lesion by a tendency to the solidification of the fibrin.

Lister married at the age of twenty-nine, the ceremony taking place, after Scotch fashion, in the drawing room of the Syme house. Dr. John Brown, the author of "Rab and his Friends", has put on record his opinion of the bride and bridegroom: "Lister is one who I believe will go to the very top of the profession." Remembering that the bride, when a baby, had been ill and unconscious for several days, he said: "As for Agnes, she was once in Heaven when she was a very little girl, and she has borne the marks of it ever since." Thus began the happiest of unions, which was to last for nearly forty years. Lister owed much to his wife, and a large amount of his work is recorded in her handwriting. On returning from a honeymoon on the Continent, Lister was unanimously elected to the staff of the Royal Infirmary in Edinburgh, a chance vacancy having arisen owing to the death of one of its surgeons in the Crimea.

The Third Phase.

He had hardly been three years in practice when he was faced with a difficult choice. The Regius Professorship of Surgery at Glasgow was vacant, and he was approached. This was a compliment indeed, and showed the high professional esteem in which he was held long before he gained fame by his introduction of antiseptic surgery. He was led to believe that, coincidently with his appointment to the professorship, he would secure a place on the

staff of the Glasgow Infirmary. It will, I think, be recognized that at such a time there was every reason to remain in Edinburgh. Reluctantly he let his name go forward, and in January, 1860, he received the appointment at the hands of the Crown. The staff appointment was in the hands of the local board, and it is hardly believable that when he urged the importance of his having beds, not only for his own sake, but for that of his pupils, he was told with dignity that his request was an Edinburgh idea and that the Glasgow Infirmary was a curative institution and not an educational one. After being rebuffed for a year, Lister's persistence met with its reward. With his advent to Glasgow "began the decade that was to alter the face of surgery".

Lister had no reason to complain of his reception by his new colleagues at Glasgow, among whom was the future Lord Kelvin, the Professor of Physics. At the early age of thirty-three he joined his father as a Fellow of the Royal Society. Such a distinction testifies to the high competence of the man five years before he was to enter upon the research which made him famous.

His family vies with that of the contemporary Darwins in the harvesting of honours, for the same distinction soon came to his brother Arthur and to one of Arthur's sons in due course. In addition, two other grandsons were to receive titles for their distinguished work in surgery.

On the Glasgow, and most historic, phase of Lister's life I do not propose to tarry. At the outset of his stay there, for all his skill and ability, the wounds still suppurated as they did when Shakespeare wrote: "What wound did ever heal but by degrees"; and gangrene and blood poisoning were rife in his wards. In "Rab and his Friends" what so often happened after an operation is beautifully described:

For some days Ailie did well . . . but four days after the operation my patient had a sudden and long shivering, a "groosin", as she called it. I saw her soon after. Her eyes were too bright, her cheek coloured; she was restless and ashamed of being so; mischief had begun. On looking at the wound, a blush of red told the secret; her pulse was rapid, her breathing anxious and quick . . . . Ailie got worse; began to wander in her mind, gently; then delirium set in, strong, without pause . . .

The intellectual power, through words and things,
Went sounding on, a dim and perilous way.

Finally, the passing of the delirium, exhaustion and the end.

Lister, amid these gruesome happenings, had, however, already satisfied himself and taught his students that pus in a wound meant decomposition of tissue, that this was somehow dependent upon but not due to the air. He now divined from Pasteur's work that the microorganisms were the enemy.

Those viewless beings
Whose mansion is the smallest particle
Of the impassive atmosphere.

To attack them in their citadel, the wound, was not enough. They must be destroyed before they entered the gates. Before leaving Lister joined in battle with the enemy, let me display to you the picture Watson Cheyne's fancy has limned of his chief:

I like to think of Lister, with his courtly manners and indomitable courage, as one of the knights of olden times sallying out single-handed to find and destroy a formidable enemy, armed with a great working hypothesis and a bottle of carbolic acid. He had never seen his enemy, but he saw his ravages on every hand; he did not know how he acted in producing diseases; he did not know where he lived or how he lived. He knew nothing about toxins or antitoxins, opsonins, agglutinins and the like. All he knew was that the enemy was probably a tiny unicellular body belonging to the vegetable kingdom. All he cared for or needed to know was that he had a terrible enemy who, however, had his vulnerable point, and that the gain was so great, if the battle were won, that it was well worth while to devote his whole mind, and his whole energies, and his whole life to the solution of the problem.

I now leave Lister engaged with the enemy, and resume the story three years later.

The victory has been won, because Lister, and Lister alone, understood the conditions necessary to victory. We find him insisting modestly at a meeting of the Medico-Chirurgical Society of Glasgow on the magnitude of the victory and showing how the spoils might be gathered. "In order", he declares, "that the antiseptic system of treatment may confer upon mankind all the benefits of which it is capable, three things appear to be indispensably requisite. First, that every surgeon should be convinced of the reality and greatness of those benefits, so that he may be induced to devote to the antiseptic dressing of a case the same kind of thought and pains as he now, if at all worthy of the name of surgeon, bestows upon the planning and execution of an operation; second, that these efforts on his part should be directed on sound principles; and, thirdly, that for carrying out these principles he should have thoroughly trustworthy practical means at his disposal".

Let it not be thought that Lister was the first to use antiseptics or even carbolic acid, which had been discovered as long ago as 1834. But there was a great gulf fixed between those who used antiseptics in the treatment of wounds and Lister's use of them. They used them empirically, much in the same way as an ignorant housewife of today might splash phenol over a smelly drain. Lister had reason for the faith that was in him. He writes:

In pursuing this object (that is, the prevention of decomposition and suppuration in wounds) we are guided by the germ theory, which supplies us with a knowledge of the nature and habits of the subtle foe we have to contend with, and without a firm belief in the truth of that theory, perplexity and blunders must be of frequent occurrence. The facts upon which it is based appear sufficiently convincing.

There were two men besides Lister who realized the great importance of Pasteur's work on the cause of putrefaction, but had not the vision to apply it—Jules Lemaire and Spencer Wells. The former, as early as 1859, washed his wounds with various antiseptics, notably carbolic acid, but he never suggested that antiseptics should be used, as Lister used them, to kill the germs before they gained a footing in the body or the wound. A few years later, in 1864, Spencer Wells, a great practical surgeon and a bold operator, and subsequently President of the Royal College of Surgeons of England, in an

address at the annual meeting of the British Medical Association, gave a most excellent description of Pasteur's researches and their bearing on surgery. But he did not follow up the logical conclusions that he drew from Pasteur's work.

No greater proof of Lister's confidence in his own methods can be adduced than the story of the operation on his sister Bella:

> She came to him with a growth that could only be removed by a severe and extensive operation. If left alone it was bound to be fatal. But under the usual conditions an operation would have been no less so. Both Paget in London and Syme in Edinburgh had considered it unjustifiable. Lister was sure that his methods could make it safe. And so it proved, the growth being removed without a trace of secondary infection.

The Fourth Phase.

After ten years in Glasgow the scene is laid in Edinburgh, where Lister had succeeded Syme as professor of clinical surgery. Over a hundred Edinburgh students had signed a letter begging him to apply for the post, "since your method of antiseptic treatment constitutes a well marked epoch in the history of British surgery and will result in glory to the profession and unspeakable benefit to mankind".

Lister was now the leading surgeon north of the Tweed, and had the distinction of operating on Queen Victoria for an axillary abscess. It was in Edinburgh that he developed the spray, the antiseptic gauze dressing and a new use for Chassaignac's rubber drainage tube. Lister had great moral courage, and even a court appointment could not undermine it. Queen Victoria, influenced by the Stephen Coleridges of the day, once asked him to take action for the suppression of experiments on animals. He declined an invitation he could not conscientiously accept; for, as he wrote, he "was persuaded that his doing so would not promote the real good of the community, which he knew to be Her Majesty's only object in the matter".

Lister's personality, as it appeared to W. E. Henley, who was his patient in the Edinburgh Royal Infirmary, is preserved for us in a fine poem. I invite you to examine the copy before you of his portrait in the Royal College of Surgeons of England, whilst I read the lines and see whether you agree with the poet.

> His brow spreads large and placid and his eye
> Is deep and bright, with steady looks that still.
> Soft lines of tranquil thought his face fulfil,
> His face at once benign and proud and shy.
> If envy scout, if ignorance deny,
> His faultless patience, his unyielding skill
> Innumerable gratitudes reply.
> His wise rare smile is sweet with certainties,
> And seems in all his patients to compel
> Such love and faith as failure cannot quell.

Lister's practice was never so large and he was never so beloved as during the seven years of his second sojourn in Edinburgh, though even there his discovery met with much contemptuous criticism. For many years Lister was to find that it was easier to kill microbes than to convince men.

The Fifth Phase.

Whose inspiration it was to invite Lister to fill the chair of clinical surgery at King's College in succession to the brilliant Sir William Fergusson I cannot say. Support of the proposal was by no means unanimous. Ultimately the chair was given to another, and Lister, to the profound distress of Edinburgh, agreed to accept a position that had been specially created for him.

He turned his back on Scotland and met with a reception at King's even more chilly than he had imagined. A nursing sisterhood then held sway there and offered passive resistance to his "new-fangled ideas". He missed, too, the hundreds of enthusiastic students whose idol he was at Edinburgh. It is almost incredible, and yet it is a fact, that Lister for some time had difficulty in filling the few beds that had been allotted to him. Not that this paucity would greatly trouble him, for he held most strongly that one individual case, if it shows new pathological facts, is worth as much as a million.

Lister had hardly settled down at King's when, to the astonishment of his colleagues, he proceeded to cut down upon a broken knee-cap and wire the two fragments together. For London, it was an unheard-of operation. "When this poor fellow dies", said a scandalized surgeon, "someone ought to proceed against that man for malpraxis." But the "poor fellow" recovered with a perfect limb. Sir Charles Ballance was present at that operation and was also present at a meeting of the Medical Society of London when Lister described the operation and showed the patients on whom the operation had been done. One of the senior surgeons, on viewing them, exclaimed: *"C'est magnifique, mais ce n'est pas la chirurgie."* The scoffers smiled at his theory, so that their attitude of mind can be understood.

In 1879 a notable discussion on antiseptic surgery took place at Saint Thomas's Hospital, in the course of which Lister's veracity was impugned. In reply, he felt, he said, that there was something more congenial and more profitable than to compile statistics. In a great hospital he had had only one case of pyæmia in five years. It was wished, he said, that he should state that he had had so many amputations with no pyæmia, so many excisions of the mamma with no pyæmia, and so forth. He went on to say:

> I confess I do not see any reason for this desire; for if I could be supposed guilty of making a false statement, of having had only one case of pyæmia in 5 years in 60 beds under my charge, I should be thought equally capable of compiling a false table of statistics.

Later on in his reply he quotes a weighty statement attributed to Morgagni: *"Perpendendæ non numerandæ observationes"*, which may be rendered in English: "Cases should be pondered, not numbered." He concluded a forcible reply as follows:

> In such a gathering as I see before me I cannot avoid speaking warmly on a matter so near to my heart. I have been charged with enthusiasm, but I regard

enthusiasm with reference to the avoidance of death, pain and calamity to our fellow creatures as a thing not at all to be ashamed of, for I feel this to be a matter of which I may say, in the words of Horace:

Æque pauperibus prodest, locupletibus æque
Æque neglectum pueris senibusque nocebit.

And he might have added the words of Harvey:

The die is cast and my trust is in the love of truth; and the candour that inheres in cultivated minds.

And so it came to pass that those who knew not Joseph learned wisdom or were displaced by those who did.

I have known two eminent disciples of Lister, Sir Charles Ballance and Hamilton Russell, each of whom was proud to acknowledge the inspiration he had derived from the master surgeon.

Sir Charles Ballance has confided that the contact with Lister which he most highly prized was an occasional invitation to visit him in his study in Park Crescent.

He received me with delightful old world courtesy and a wise, rare smile of welcome, and then said in his simple way: "Tell me what you have been doing. Have you any microscopic slides to show me?"

He would then talk to Ballance about his investigations for an hour or more.

I cannot refrain from expressing in this place the admiration I feel for Sir Charles Ballance himself and his work. He is a true disciple of Harvey and of Lister, in that he "studies and seeks out the secrets of nature by way of experiment", and this he has done throughout his professional life. Sorely stricken though he has been in many ways, his head remains unbowed, and rest from research work is not yet his desire. I have his permission to refer to some of his recent labours and the results of his experiments. These he brought before the Royal College of Surgeons of England in the Lister Memorial Lecture which he delivered last year. After his return from Australia a few years ago Sir Charles received an invitation from Arthur B. Duel, in the United States of America, asking him to undertake certain investigations. Duel is a man of amazing energy and enthusiasm, and very skilled in delicate surgical manipulations. Sir Charles and Duel jointly undertook an investigation on the subject of nerve grafting in the Fallopian aqueduct of baboons. Sir Charles performed the first operation for the relief of facial paralysis in 1895. He now, as the result of his experience in operating on baboons, makes the following statement:

I wish definitely to state that there is now only one operation for facial palsy, and that is the nerve graft operation in the Fallopian aqueduct. I have no shadow of doubt that in twenty years from now the operation will be definitely established in every surgical clinic as the operation of choice for facial palsy.

Owing to the financial blizzard which struck the land of freedom, Sir Charles had, two years ago, to return to London, where he now spends his time working in the new laboratories of the College of Surgeons. Some months after his departure Duel wrote to him to say that a degenerated nerve graft, that is, a nerve graft taken from a nerve that has been cut off from its central connexions for from eight to thirty days, will cause recovery of function in the paralysed muscles in half the time that is required when a fresh graft is employed. Sir Charles remarks that, in his view, this discovery is of much surgical importance and is of universal application wherever in the body a nerve graft has to be used.

In the coming time surgeons must renounce the use of fresh nerve grafts; the rule is absolute that a degenerated nerve graft must be employed.

This emphatic statement by so distinguished a disciple of Lister demands our attention. Sir Charles, in his address, pronounces an eloquent panegyric on Lister. Its substance is taken from Thucydides:

History has deliberately pronounced that among eminent men no one has left a more stainless and none a more splendid name. The whole earth is the sepulchre of Lister. In this land and on far-off shores there is an abiding memorial; that best of all memorials begot in the ventricle of memory. No pen or chisel has traced it; it is graven not on marble or brass, but in the hearts of men.

And he pictures every operating theatre in the world as a temple in which the spirit of Lister dwells.

Hamilton Russell displays the same reverence for the maker of modern surgery. He attributes his own elevation to the presidency of the Surgical Section of the Australasian Medical Congress to be the direct result of the teaching of Lister; and he rejoices in the knowledge that whatever has been found worthy in him may be ascribed to Lister's influence, his teaching and his example. "In accepting the honour", he said, "I would offer it as the only tribute it is in my power to offer to his beloved and honoured memory." Towards the end of his address he says:

Those who knew Lister intimately, who became conscious of his humble-mindedness and modesty, his purity in thought and deed, his deep and child-like religious faith, and his abounding love of his fellow man, recognize that they have witnessed the culmination of the highest qualities that civilization has yet brought forth.

May I, in conclusion, imitate Watson Cheyne and indulge in a flight of fancy by likening Lister to Perseus. Many of you will remember the story of Perseus and the seeming impossibility of his undertaking to bring to King Polydectes the head of Medusa, the only Gorgon subject to mortality, and how in his quest he was patronized by the gods. Pluto lent him his helmet, which made the bearer invisible; Minerva, her buckler, which was as resplendent as glass; from Mercury, wings and the talaria with a short dagger made of diamonds. So, too, did Divine Providence bless Lister with the possession of three advantages which combined to make him so powerful an advocate of antiseptic surgery. He had the professional position which gave him the authority; he had the gifts of fortune which gave him the means; and, lastly, he had the

lavish gifts of Nature which gave him in happy combination

> The patient thought, the steadfast will,
> Resolve and foresight, strength and skill,

which he has laid upon the altar of suffering humanity.

Fifteen months ago, in the Loggia de Lanzi, at Florence, I was privileged to see Cellini's beautiful statue of Perseus holding the bleeding head of Medusa, the Gorgon he had destroyed. That great work of art seems to my mind to be symbolical of Lister's struggle with infection and of his achievement, and in my fancy I grave on it the words of England's greatest immortal in letters:

> I will through and through
> Cleanse the foul body of the infected world.

Papers Published by Lister Prior to 1867, the Year of his First Paper on Antisepsis.

"Observations on the Contractile Tissue of the Iris", 1853.
"Observations on the Muscular Tissue of the Skin", 1853.
"On the Minute Structure of Involuntary Muscular Fibre", 1857.
"Report of Some Cases of Articular Disease Occurring in Mr. Syme's Practice, Exemplifying the Advantages of the Actual Cautery", 1854.
"On the Flow of Lacteal Fluid in the Mesentery of the Mouse", 1857.
"An Inquiry Regarding the Parts of the Nervous System which Regulate the Contractions of the Arteries", 1857.
"On the Cutaneous Pigmentary System of the Frog", 1857.
"On Spontaneous Gangrene from Arteritis and the Causes of Coagulation of the Blood in Diseases of the Blood Vessels", 1858.
"A Case of Ligature of the Brachial Artery illustrating the Persistent Vitality of the Tissues", 1858.
"Preliminary Account of an Enquiry into the Functions of the Visceral Nerves, with Special Reference to the So-Called Inhibitory System", 1858.
"Some Observations on the Structure of Nerve Fibres", 1859.
"Notice of Further Researches on the Coagulation of the Blood".
"On the Coagulation of the Blood", 1863.
"On Anæsthetics", 1863.
"Notes on the Examination of an Exostosis Removed by Mr. Syme from the Os Humeri of a Young Lady aged about 20 Years", 1854.
"Report of a Case of Carbuncle Occurring in Mr. Syme's Practice, illustrating especially the Pathology of that Disease", 1854.
"On the Early Stages of Inflammation", 1858.

Acknowledgements.

In the preparation of this address I owe much to: "The Life of Lord Lister" (Rickman Godlee), "Lister and his Achievement" (Watson Cheyne), "A Harley Street Calendar" (H. H. Bashford).

THE X RAY EXAMINATION OF THE APPENDIX.

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Recently there has been a considerable amount of comment in the literature in regard to the vermiform appendix, from both the clinical and the surgical standpoint, and possibly aroused by the publicity given to it in the lay Press. It would be well to review the appendix from the radiological standpoint and to discuss briefly the various signs which the radiologist may encounter during the examination for a suggested chronic appendicitis.

It may be stated here that there is no one sign pathognomonic of chronic appendicitis and that the diagnosis must be based on the association of several observations recorded during the examination under the fluorescent screen. It is partly for this reason that I have undertaken this short *résumé*, for the radiologist is sometimes called upon to hazard an opinion on X ray films of the caecal region alone and has not the added and all-important information gleaned by actual palpation during screen examination.

It has been stated[1] that few normal appendices have been found by surgeons and pathologists and that the diagnosis of appendicitis upon any grounds whatever is not at all hazardous, provided the novice make sure that the appendix has not previously been removed.

The first part of the above statement may be a compliment to the clinicians of the past, but the latter part appears to be an admission of failure on the part of the radiologists to comprehend the true significance of many signs which are now considered to be normal physiological phenomena. For instance, if contrast meal entered the appendix it was considered by some authorities as being a sign of pathological change, whereas others considered that the converse was equally suggestive. The true significance of these findings will be discussed later.

For obvious reasons the radiologist seldom carries out an examination in acute appendicitis, and his observations are confined to the subacute and chronic types of inflammation.

The method of examination is by the ingested twenty-four hour barium meal—usually the day following the examination of the stomach and duodenum, when the meal has passed naturally into the colon. Even though it is only the appendix that is to be examined, it is well to screen the stomach and duodenum whilst the patient is taking the meal, for often it is difficult to differentiate clinically between duodenal ulcer and the reflex spasm of the duodenum produced by peritoneal irritation from a chronic appendicitis. This relation of reflex peritoneal irritation of the duodenal bulb is a well known and established fact and is observed in any peritoneal irritation elsewhere in the abdomen.

One and a half pints of barium meal, made up in the usual manner, are given eighteen to twenty-four hours before the examination. The addition of two fluid ounces of "Petrolagar" improves the consistency of the meal and assists its subsequent passage through the colon. After taking the meal the patient carries on the usual mode of life, with the exception that no aperient is allowed. The following morning the examination is carried out under the fluorescent screen.

The patient lies in the supine position (the X ray tube is under the table and the fluorescent screen is over the abdomen), so that palpation of the caecum and caecal region can be adequately performed and the area at the same time thoroughly examined by rotation of the patient into the oblique positions and the observations fully made. A pillow under the hips of the patient greatly assists palpation. The erect position of the patient should never be employed, for the reason that the colon tends to fall, carrying the caecum out of reach of palpation into the bony pelvis and the overlying coils of barium-filled bowel obscure a clear view. The barium enema is never employed for the examination of the appendix, as the latter is rarely outlined by this method in the examination of the colon as a whole.

In approximately 70% of cases the appendix will be outlined by the ingested meal method, provided subsequent examinations at intervals of a few hours are made should the appendix not be filled at the first screening. The physiological process of filling and emptying of the appendix has been noted whilst the caecum remains filled with barium, and it may be that the appendix is empty and therefore not outlined at the first examination, but will subsequently fill in the course of a few hours.

Thus there is no optimum time for the examination, but the appendix may reasonably be expected to fill within sixteen to twenty-four hours after the ingestion of the meal. I have noted the appendix clearly outlined at eighteen to twenty hours which had emptied at a subsequent examination at twenty-four hours. The converse also has been noted on numerous occasions. The anatomy and physiology of the appendix is well known, but it will suffice to say that the size and shape vary considerably. The average length is approximately seven centimetres, comma or S-shaped, and lying medially and below the caecum. The shape of the normal appendix may be seen to alter gradually owing to the intrinsic muscular contractions that occur slowly in the caecal region.

The ingested meal rapidly reaches the caecum, the process usually taking from two to three hours, but the appendix does not become visible for some hours after the caecum and ascending colon have filled. Physiologically the normal appendix fills and empties slowly and at varying intervals of time, while the caecum remains filled. This process is said to commence at about the sixth to eighth hour after ingestion, but the reason that the appendix does not become visible to the radiologist at so early a stage is possibly explained by the fact that the normal appendix may be full of previously ingested food residues and must empty before barium can subsequently enter and reveal its relative size, shape and position.

Just as its size may vary, so also may its position vary between very wide limits, and it is interesting to note how often its position does not correspond with McBurney's point. The appendix has been reported from time to time by various authors to occupy practically any position within the abdomen from the left iliac fossa to the peritoneal sac of a right inguinal hernia; and I have seen films demonstrating the caecum and appendix situated under the liver and within an umbilical hernia. The latter was a classical example in the collection of the Royal Infirmary of Edinburgh.

Certain positions, however, are regarded as one point towards the diagnosis of associated pathological change, particularly the retrocaecal position, for if the appendix happens to fill with contrast meal and be demonstrated in one of these positions, it is a considerable aid to the surgeon in his method of approach.

Now what are the signs observed by a radiologist during the screen examination and palpation, and what significance can be drawn from each to lead to the ultimate diagnosis?

The first of these is whether the appendix is visible or not. If it is not filled with barium at the time of examination, a further examination several hours later may reveal it well outlined. For this reason non-visualization of itself is definitely not a sign of pathological change, though some writers in the past maintained that it was a sign. If, however, there is persistent non-filling, together with other suggestive or corroborative signs, then it is of definite significance. The associated signs may be direct caecal tenderness or spasm, some degree of caecal fixation, and ileal or caecal stasis.

Persistent non-filling associated with some or all of the above additional signs would suggest that inflammatory swelling or adhesions from chronic inflammation had occluded the lumen or caused fibrosis of the musculature, preventing the normal physiological function.

The question of previous operation must always be raised where there is persistent non-filling, for adhesions and tenderness on palpation may, and often do, exist after appendicectomy. Mistakes in this regard are more than possible when the radiologist is asked to express an opinion upon a film alone and practically no history is given.

Thus it will be seen that if the appendix is not definitely outlined at the first or subsequent examination within a few hours later, it must be regarded as a suggestive sign of pathological change, but is conclusive where definite caecal tenderness, spasm, fixation *et cetera* are superadded.

The following remarks refer to the appendix which does fill and therefore becomes visible and is palpable under the fluorescent screen. If the appendix is visible, is regular in outline, and the caecum and appendix are freely movable and, above all, not tender on direct palpation, then there is every reason to presume that there is no associated pathological change. The normal appendix also completely empties as the meal passes onwards from the caecum.

The length varies considerably, and it is usually the long appendix which takes a considerable time to expel the barium meal after the caecum has emptied. Indeed, barium remnants have remained

for days even after severe purgation. The long appendix, for this reason, should be regarded with suspicion, but only if it remains filled or partly filled, for it is reasonable to regard a degree of stagnation of bowel contents as a predisposing cause of inflammation or as a loss of the physiological power of contraction due to fibrosis from preexisting chronic inflammation.

The normal shape is usually comma or S-shaped, but true or apparent kinks often occur. Apparent kinks are merely a foreshortening of the shadow of a normal curve, and the normal curve is demonstrated on rotation of the patient into another plane. True kinks may be due to a short mesentery or to adhesions, and if constant despite palpation and if associated with local tenderness, they are a suggestive sign of pathological change. As before stated, the appendix may be anywhere in the abdominal cavity or peritoneal sacs associated therewith, but the usual position is in the right iliac fossa, below and medial to the caecum.

Variations from the normal position are either congenital or acquired, the latter usually resulting from adhesions drawing the caecum out of its usual position and carrying with it the appendix. Slight variations from the normal position which are considered to be associated with chronic inflammation are the retrocaecal and the right lateral positions. The former is best demonstrated when the caecum is partly empty, otherwise the dense barium shadow of the full caecum obscures the position of the appendix behind it, and it is often difficult to distinguish the appendix posteriorly, even when the patient is rotated into a lateral position.

The normal appendix should be freely movable and not tender on palpation. A short mesentery will restrict free movement, but in the absence of definite tenderness or other sign this restricted movement is of little significance. If it is fixed in position due to adhesions, efforts to separate its shadow from the caecum or adjacent viscera will fail and the adjacent portions of caecum or colon will move as a whole with it, and the movement will be accompanied by local tenderness.

Additional signs will be an irregular outline and possibly constant kinks. Adhesions to the tip of the appendix only may be present. Here the appendix is movable, with the exception of the tip, and during palpation the appendiceal shadow may elongate and return to its former position. Constant strictures of its lumen may also be noted. Adhesions in the caecal region are manifested by fixation, irregularity in outline, ileo-caecal stasis, and associated pain on palpation.

Fæcoliths may be found associated with constant irregularity of the lumen. These may be calcified and in a plain skiagram may simulate a right ureteric calculus. If the appendix is outlined sufficiently well, these concretions appear as rounded or ovoid areas of less opaque substance, usually towards the tip of an appendix with an irregular lumen. Foreign bodies may sometimes be detected.

One case I recall was that of a patient who was suspected of having a chronic appendicitis; when he was examined it was discovered that several small shot had lodged in the appendix and had presumably been there for some considerable time, as he had not eaten game for some months. They were present at operation in a chronically inflamed and fibrosed appendix.

Incomplete evacuation of barium residues from its lumen some hours or even days after the caecum and bowel have emptied indicates a definite chronic fibrosis of its walls and is not necessarily associated with the long appendix, as above mentioned.

Caecal stasis and ileo-caecal or ileal stasis may be a further sign. The normal evacuation time for various types of colon varies considerably, but as a rule the colonic content moves evenly onwards to the distal part of the hepatic flexure. True caecal stasis is shown when the caecum remains partly filled and the remainder of the colon, except the sigmoid and rectum, is practically empty.

Portion of the opaque meal may still be present in the terminal ileum at twenty-four hours or longer after the ingestion of the meal, and this usually indicates the presence of adhesions in the region of the ileo-caecal valve. The thin wisps of meal in the terminal part of the ileum can be mistaken for the appendix unless careful examination is made and technically good X ray films are obtained. Ileal stasis alone only suggests pathological change, for there are other causes than chronic appendicitis. The same applies to caecal and colonic spasm.

A further sign which has been quoted in the past as suggestive of pathological change is insufficiency of the ileo-caecal valve observed in colonic examinations by the barium enema. This phenomenon is of no pathological significance. In fact it is the degree of filling which the radiologist tries by palpation to obtain in all enema examinations of the colon, for he then knows that filling has been complete. Even with such filling it is a rarity to outline the appendix, which is a cul-de-sac. Insufficiency of the ileo-caecal valve is seen in cases in which the appendix has been previously removed, and also in cases in which there is no clinical sign or suggestion of chronic appendicitis.

I have purposely left the sign of pain on direct pressure over the caecum or appendix as the last consideration, for it may be quite definitely stated that most of the other signs lose a great deal of their conclusiveness without the accompanying sign of pressure pain on palpation. This sign is never absent when several of the other signs previously mentioned definitely exist, but it is the most significant one when the other signs are suggested but not conclusively proven to be present.

It may be quite true that "few normal appendices have been found by surgeons and pathologists", as above mentioned, but from my previous remarks it will be seen that a little more than "any grounds whatever" is required for a correct diagnosis of chronic appendicitis, especially when the radiologist is called upon to assist in the elucidation of vague abdominal symptoms which clinically may only sug-

gest appendicitis and which might quite easily be due to chronic cholecystitis, duodenal ulcer or visceroptosis *et cetera*.

Recently I carried out a Graham's test of the gall-bladder in a girl of sixteen years who had had a "chronic appendix" removed some twelve months previously. Her gall-bladder contained a number of transradiant calculi.

Cholecystitis and appendicitis resemble one another clinically, and the fact that cholecystitis is a disease of early life, with later manifestations, is often forgotten.

Finally, an appendix which fills and empties satisfactorily and which is freely movable and not tender on direct palpation, must be considered to be normal, even though caecal spasm, irregular lumen, and other minor signs mentioned above, may also be present at the time of examination.

Reference.

(a) R. D. Carman and A. Miller: "Diseases of the Alimentary Canal", 1917, page 517.

A CONTRIBUTION TO THE STUDY OF THE HEREDITY OF BLOOD GROUPS IN AUSTRALIAN FAMILIES.

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OWING to the importance of the theory of the heredity of blood groups in medico-legal work, some figures are here presented for the population of Sydney.

Material for this work was obtained mostly by the examination of families consisting of father, mother and child, through Saint Margaret's Hospital. A few were obtained from adult families either in private or hospital practice, as opportunity offered.

Nomenclature.

The classification of the blood groups according to Jansky has the right of priority, and in 1921 *The Journal of the American Medical Association*[1] adopted this classification and recommended it for universal use. However, many institutions and countries continued to use the classification according to Moss, and this has been the source of endless confusion. In order to remedy this, the properly qualified international authority, namely, the Commission for the Standardization of Sera of the Public Health Committee of the League of Nations, has entirely done away with numerals and has adopted an alphabetical terminology corresponding to the agglutinogen content of each group, that is, O, A, B, A B.[2][3] In 1927[4] the American Association of Immunologists adopted this classification, which has now been widely accepted throughout the world. Its utility is obvious, as all sources of confusion which may be exceedingly dangerous are eliminated, it has a more scientific basis, teaching is simplified, and, most important of all, it facilitates communication between workers in different countries.[5]

This terminology should now be adopted by all hospitals, blood donors' associations and medical schools throughout this country and the old terminologies of Moss and Jansky should be discarded.

The following shows the relation between the old and new terminologies:

Group O corresponds to Jansky I and Moss IV. No agglutinogens are present in the cells, but both agglutinins anti-A and anti-B are present in the serum.

Group A corresponds to Jansky II and Moss II. The cells of this group contain the agglutinogen A, and the serum the anti-B agglutinin.

Group B corresponds to Jansky III and Moss III. The cells of this group contain the agglutinogen B, and the serum the anti-A agglutinin.

Group A B corresponds to Jansky IV and Moss I. The cells of this group contain both agglutinogens, but the sera does not contain either of the agglutinins.

The question of sub-groups containing additional agglutinogens and agglutinins, as described by Guthrie and Huck[6] and Simson[7] and other workers, will not be discussed here. Suffice it to say that in all the blood of adults in this series of examinations no expected reaction failed to occur and no unexpected reaction took place between specimens of blood of the same group. It still remains to be proved whether the sub-groups described are true sub-groups or whether the reactions are due to quantitative differences.

The agglutinogen is contained in the stroma of the red cells,[8] and Halleur[9] has shown that it is soluble in water and that extracts of the stromata, and also water in which erythrocytes have been washed, contain the agglutinogen. With some erythrocytes the agglutinogen was more soluble than with others. The chemical composition of the agglutinogen is complex.[10][11] These individual characters of the blood, where they are easily demonstrable, have been shown to be only a special example of properties which all the cells of the body possess, and the presence of these group-specific antigens has been demonstrated in various kinds of fixed somatic cells.[12]

The isoagglutinins present in the blood serum can be demonstrated in the breast milk of nursing women,[13] in pathological fluids as transudates and exudates (example, Family 9).

According to Rona and Krebs[14] and Bleyer,[15] isoagglutinins are linked with the euglobulin fraction of the serum and only traces are present in the pseudoglobulin fraction.

Toda[16] has demonstrated that the platelets of human blood are not able to be relegated to the four groups by means of typing sera. Some work by Doan[17] suggests that leucocytes possess individuality and that there is a definite incompatibility between the blood plasma of some individuals and the white blood cells of others.

Constancy of the Blood Group in an Individual.

The blood group of an individual is an immutable constitutional characteristic. It remains unchanged throughout life and can be altered neither by disease, medicaments[18] nor blood transfusions.[19] Variations may occur, and have frequently been reported, in the strength of the agglutinogens and titres of the agglutinins. Harper and Byron[20] have noted the effect of diet on isohæmagglutinins, especially amongst individuals belonging to Group B.

Edgecombe[21] tested the blood serum of eighteen women at various periods of pregnancy and after delivery and found that the fœtus had a definite influence on the titre of the mother blood, most marked with a fœtus of a group different from the mother, where the rise in agglutinating titre of the cells averaged 83%, and of the serum 330%.

Hübener[22] reports an instance in which the expected agglutinogen A could be demonstrated only after recovery from illness. These variations in titre of serum and variations in the agglutinability of the cells, together with pseudo-agglutination, which is especially apt to occur in disease, account for the erroneous conclusions drawn by a few workers.

The work of Huck and Peyton[23] demonstrated that there is no change of blood groups after ether anæsthesia, which was claimed by Levine and Segall.[24] Thomsen and Kettel[25] have obtained results on a large amount of material and find that agglutinins vary in their titre at different ages, and in old age the titre gradually lessens until it can fall as low as in infancy, a process of serological involution. The agglutinogens develop early in fœtal life and have been described in the blood of fœtuses of four, five and six months;[26][27][28] Jones[29] demonstrated them in a seven months fœtus. Karshner's findings[30] confirm the observations of de Biasi[31] that the corpuscles of new-born infants have their full quota of receptors (agglutinogens) and that the distribution of blood groups in infants is the same as in adults.

A few cases are on record in which delay in the maturation of the agglutinogen has occurred.[32] Smith[13] reports two cases in which an O group mother had passed on her agglutinins to her new-born infants and the cells of the umbilical blood were inagglutinable. In a few days, however, agglutinogens were present in the blood of the infants, who belonged to Groups A and B.

These two cases are of great importance and show that umbilical blood should never be used for forensic examinations. According to Thomsen and Kettel, agglutinogens also undergo a process of maturation and the titre rises slowly up to the age of fifteen to twenty years.

The agglutinins are much later in developing than the agglutinogens. Happ showed that at birth and during the first month of life isoagglutination is rarely present; but the percentage of infants in whom the isoagglutinin group is established increased with age, so that after one year the group is usually established, and after two years is always present, as in adults. Jones,[29] by using special methods, was able to demonstrate agglutinins in a much larger percentage of cases than Happ, and concluded that 100% of new-born infants can be correctly grouped by means of their cells.

The appearance of the agglutinins in infants can be hastened by certain stimuli, as blood transfusion.[33]

Sussman[34] states that the new-born child inherits group-specific agglutinogens, but not iso-agglutinins, and that the inhalation of particles of foreign saliva suspended in the air gives rise to an immunological formation of isoagglutinins directed against the agglutinogens which the child does not possess. This process is gradual and presumably must vary with the population of the district.

In the series of infants all between seven and nine days old, which form the large part of the material for this paper, it was the exception rather than the rule to be able to demonstrate agglutinins by the ordinary method.

Heredity of the Blood Groups.

In this discussion it is presumed that the reader possesses a knowledge of the Mendelian theory of heredity.

In 1910 von Dungern and Hirszfeld published the first systematic work on the inheritance of the blood groups and brought forward their theory of inheritance in agreement with orthodox Mendelian principles regarding A and B as dominants. According to this theory, the blood group of an individual is the result of four characters united in two allelomorphic pairs, A and not-A, B and not-B, completely independent of each other and situated far apart in the chromosome system. The four blood groups can be formed by the combination of the two pairs of allelomorphs, each pair giving rise to three genotypes and two phenotypes. For example, for the pair A and not-A the following would hold:

A A, dominant homozygote
A, not-A, dominant heterozygote } Phenotype A.
Not-A, Not-A = recessive homozygote.

And the same for B.

The combination of the two pairs therefore gives rise to nine genotypes and four phenotypes.

Table I.

Blood Groups and Hereditary Types (after Hirszfeld).[18][20][27]

Phenotypical Groups.	Genotypes.			
O ab	Not-A, Not-A. Not-B, Not-B pure.			
A b	A A. Not-B, not-B pure.	A, not-A. Not-B, not-B hybrid.		
B a	Not-A, not-A. B B pure.	Not-A, not-A. B, not-B hybrid.		
AB o	A A. B B pure.	A, not-A. B, not-B di-hybrid.	A, not-A. B B mono-hybrid.	A A. B not-B mono-hybrid.

In 1924 the mathematician Bernstein pointed out that this theory was not compatible with deductions concerning random mating in mixed populations. Bernstein[10] claims that the hypothesis of three allelomorphic genes, A, B and R (O), one of which is present in each member of a certain pair of chromosomes, A and B being dominant over R (O), agrees more closely with observed facts. Since each somatic cell possesses two of these gametes—one from the father and one from the mother—the following genotypes and phenotypes occur (Table II).

Table II.

Genotype.	Phenotype.
RR (OO)	O
AA }	A
AR }	
BB }	B
BR }	
AB	AB

On the basis of elaborate analyses of available data by geneticists Bernstein's hypothesis may now be regarded as a satisfactory account of the inheritance of the blood groups.

In comparing the two theories it is to be noted that there is no disagreement about the facts most essential for the practical application of the tests, namely, "the biochemical structures of A and B never make their appearance in the offspring unless they are present in one or both of the parents".

According to the theory of von Dungern and Hirszfeld, an O parent can have a child A B and a parent A B can have a child O; according to Bernstein's theory this is impossible.

Bernstein's theory regarding this particular point is supported by an impressively large number of observations, the exceptions occurring mostly in the older literature, where the possibility of error was great. However, excluding any doubtful reports, there is one instance reported[38] in which a mother of group A B gave birth to a child whose blood was indistinguishable from Group O. According to Landsteiner,[39] it is therefore wisest to adopt a conservative attitude regarding the possibility of parent-child combination A B-O or O-A B. In this series it is interesting to note that the offspring in five families with one Group A B parent are all either Group A or Group B.

Further reliable work on a suitable section of the population is required finally to settle the matter.

Technique and Methods.

All the bloods were collected, labelled and examined personally. Blood was collected from the fathers when they visited their wives in the evenings, and from the mothers and babies between the sixth and ninth days of the puerperium. In no case was blood taken if either of the parents made any objection, no matter on what grounds. In the adults a few drops of blood were collected from a prick on the finger, some into 3·8% citrate solution and some into a clean, dry tube, in order to collect the serum. With the babies, blood was taken from a prick on the heel. It was not always possible to collect the blood in the post-digestive period. After collection the blood was placed on ice for twenty-four to forty-eight hours. The cells were washed once with citrate solution. This was done for two reasons: (i) the supernatant citrate solution from the babies' cells was frequently stained with bile pigment, (ii) to eliminate the possibility of the red corpuscles retaining sufficient of their own serum to interfere with the reaction.[40] The actual agglutination tests were performed on plain, clean glass slides, equal parts of serum and cell suspension being used. The cell suspension was always between 2·5% and 5·0%. The slide was oscillated and final readings were not taken until the end of fifteen minutes, but agglutination, when present, usually occurred within two or three minutes. It was noted that the A-a system agglutinated more quickly and strongly than the B-b system.

The sources of error tabulated by Ottenberg were carefully avoided.[41] As can be seen from the results, the inability to demonstrate agglutinins in babies' serum was a frequent occurrence. Whether these are entirely absent or only not demonstrable by this method is another question. It is known that agglutinins can be strengthened by alternately freezing and thawing the serum over a period of weeks, when the serum separates out into two layers, the upper colourless, like water, and not containing any agglutinin, the lower straw-coloured and containing the strengthened agglutinin. Unfortunately facilities for refrigeration were not available when this work was being done and this method could not be tried, so has had to be postponed until later, when I hope to be able to obtain another series of families and test the serum from the babies in this manner.

The agglutinogens, when present in the babies' blood, were easily demonstrated. In the cases in which neither agglutinogens nor agglutinins were present, the baby was classified as belonging to Group O.

Results of Investigation.

Altogether 81 families with 91 children, comprising 253 individuals, were examined. Of these individuals, 119 belonged to Group O, 107 belonged to Group A, 21 belonged to Group B, and 6 only belonged to Group A B.

There are no exceptions to Bernstein's law, as can be seen from Table III and results in detail of each family (Table IV).

Although the father's blood could not be obtained in the last case shown in Table IV, it is included because of the importance of the question of A B mothers.

These results agree with those obtained by previous workers in this field and are of the utmost importance from the medico-legal point of view.

No attempt has been made to review the literature, which has been adequately done by others; but some points bearing on the subject have been shortly discussed.

lavish gifts of Nature which gave him in happy combination

> The patient thought, the steadfast will,
> Resolve and foresight, strength and skill,

which he has laid upon the altar of suffering humanity.

Fifteen months ago, in the Loggia de Lanzi, at Florence, I was privileged to see Cellini's beautiful statue of Perseus holding the bleeding head of Medusa, the Gorgon he had destroyed. That great work of art seems to my mind to be symbolical of Lister's struggle with infection and of his achievement, and in my fancy I grave on it the words of England's greatest immortal in letters:

> I will through and through
> Cleanse the foul body of the infected world.

Papers Published by Lister Prior to 1867, the Year of his First Paper on Antisepsis.

"Observations on the Contractile Tissue of the Iris", 1853.
"Observations on the Muscular Tissue of the Skin", 1853.
"On the Minute Structure of Involuntary Muscular Fibre", 1857.
"Report of Some Cases of Articular Disease Occurring in Mr. Syme's Practice, Exemplifying the Advantages of the Actual Cautery", 1854.
"On the Flow of Lacteal Fluid in the Mesentery of the Mouse", 1857.
"An Inquiry Regarding the Parts of the Nervous System which Regulate the Contractions of the Arteries", 1857.
"On the Cutaneous Pigmentary System of the Frog", 1857.
"On Spontaneous Gangrene from Arteritis and the Causes of Coagulation of the Blood in Diseases of the Blood Vessels", 1858.
"A Case of Ligature of the Brachial Artery illustrating the Persistent Vitality of the Tissues", 1858.
"Preliminary Account of an Enquiry into the Functions of the Visceral Nerves, with Special Reference to the So-Called Inhibitory System", 1858.
"Some Observations on the Structure of Nerve Fibres", 1859.
"Notice of Further Researches on the Coagulation of the Blood".
"On the Coagulation of the Blood", 1863.
"On Anæsthetics", 1863.
"Notes on the Examination of an Exostosis Removed by Mr. Syme from the Os Humeri of a Young Lady aged about 20 Years", 1854.
"Report of a Case of Carbuncle Occurring in Mr. Syme's Practice, illustrating especially the Pathology of that Disease", 1854.
"On the Early Stages of Inflammation", 1858.

Acknowledgements.

In the preparation of this address I owe much to: "The Life of Lord Lister" (Rickman Godlee), "Lister and his Achievement" (Watson Cheyne), "A Harley Street Calendar" (H. H. Bashford).

THE X RAY EXAMINATION OF THE APPENDIX.

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RECENTLY there has been a considerable amount of comment in the literature in regard to the vermiform appendix, from both the clinical and the surgical standpoint, and possibly aroused by the publicity given to it in the lay Press. It would be well to review the appendix from the radiological standpoint and to discuss briefly the various signs which the radiologist may encounter during the examination for a suggested chronic appendicitis.

It may be stated here that there is no one sign pathognomonic of chronic appendicitis and that the diagnosis must be based on the association of several observations recorded during the examination under the fluorescent screen. It is partly for this reason that I have undertaken this short *résumé*, for the radiologist is sometimes called upon to hazard an opinion on X ray films of the caecal region alone and has not the added and all-important information gleaned by actual palpation during screen examination.

It has been stated[1] that few normal appendices have been found by surgeons and pathologists and that the diagnosis of appendicitis upon any grounds whatever is not at all hazardous, provided the novice make sure that the appendix has not previously been removed.

The first part of the above statement may be a compliment to the clinicians of the past, but the latter part appears to be an admission of failure on the part of the radiologists to comprehend the true significance of many signs which are now considered to be normal physiological phenomena. For instance, if contrast meal entered the appendix it was considered by some authorities as being a sign of pathological change, whereas others considered that the converse was equally suggestive. The true significance of these findings will be discussed later.

For obvious reasons the radiologist seldom carries out an examination in acute appendicitis, and his observations are confined to the subacute and chronic types of inflammation.

The method of examination is by the ingested twenty-four hour barium meal—usually the day following the examination of the stomach and duodenum, when the meal has passed naturally into the colon. Even though it is only the appendix that is to be examined, it is well to screen the stomach and duodenum whilst the patient is taking the meal, for often it is difficult to differentiate clinically between duodenal ulcer and the reflex spasm of the duodenum produced by peritoneal irritation from a chronic appendicitis. This relation of reflex peritoneal irritation of the duodenal bulb is a well known and established fact and is observed in any peritoneal irritation elsewhere in the abdomen.

One and a half pints of barium meal, made up in the usual manner, are given eighteen to twenty-four hours before the examination. The addition of two fluid ounces of "Petrolagar" improves the consistency of the meal and assists its subsequent passage through the colon. After taking the meal the patient carries on the usual mode of life, with the exception that no aperient is allowed. The following morning the examination is carried out under the fluorescent screen.

The patient lies in the supine position (the X ray tube is under the table and the fluorescent screen is over the abdomen), so that palpation of the caecum and caecal region can be adequately performed and the area at the same time thoroughly examined by rotation of the patient into the oblique positions and the observations fully made. A pillow under the hips of the patient greatly assists palpation. The erect position of the patient should never be employed, for the reason that the colon tends to fall, carrying the caecum out of reach of palpation into the bony pelvis and the overlying coils of barium-filled bowel obscure a clear view. The barium enema is never employed for the examination of the appendix, as the latter is rarely outlined by this method in the examination of the colon as a whole.

In approximately 70% of cases the appendix will be outlined by the ingested meal method, provided subsequent examinations at intervals of a few hours are made should the appendix not be filled at the first screening. The physiological process of filling and emptying of the appendix has been noted whilst the caecum remains filled with barium, and it may be that the appendix is empty and therefore not outlined at the first examination, but will subsequently fill in the course of a few hours.

Thus there is no optimum time for the examination, but the appendix may reasonably be expected to fill within sixteen to twenty-four hours after the ingestion of the meal. I have noted the appendix clearly outlined at eighteen to twenty hours which had emptied at a subsequent examination at twenty-four hours. The converse also has been noted on numerous occasions. The anatomy and physiology of the appendix is well known, but it will suffice to say that the size and shape vary considerably. The average length is approximately seven centimetres, comma or S-shaped, and lying medially and below the caecum. The shape of the normal appendix may be seen to alter gradually owing to the intrinsic muscular contractions that occur slowly in the caecal region.

The ingested meal rapidly reaches the caecum, the process usually taking from two to three hours, but the appendix does not become visible for some hours after the caecum and ascending colon have filled. Physiologically the normal appendix fills and empties slowly and at varying intervals of time, while the caecum remains filled. This process is said to commence at about the sixth to eighth hour after ingestion, but the reason that the appendix does not become visible to the radiologist at so early a stage is possibly explained by the fact that the normal appendix may be full of previously ingested food residues and must empty before barium can subsequently enter and reveal its relative size, shape and position.

Just as its size may vary, so also may its position vary between very wide limits, and it is interesting to note how often its position does not correspond with McBurney's point. The appendix has been reported from time to time by various authors to occupy practically any position within the abdomen from the left iliac fossa to the peritoneal sac of a right inguinal hernia; and I have seen films demonstrating the caecum and appendix situated under the liver and within an umbilical hernia. The latter was a classical example in the collection of the Royal Infirmary of Edinburgh.

Certain positions, however, are regarded as one point towards the diagnosis of associated pathological change, particularly the retrocaecal position, for if the appendix happens to fill with contrast meal and be demonstrated in one of these positions, it is a considerable aid to the surgeon in his method of approach.

Now what are the signs observed by a radiologist during the screen examination and palpation, and what significance can be drawn from each to lead to the ultimate diagnosis?

The first of these is whether the appendix is visible or not. If it is not filled with barium at the time of examination, a further examination several hours later may reveal it well outlined. For this reason non-visualization of itself is definitely not a sign of pathological change, though some writers in the past maintained that it was a sign. If, however, there is persistent non-filling, together with other suggestive or corroborative signs, then it is of definite significance. The associated signs may be direct caecal tenderness or spasm, some degree of caecal fixation, and ileal or caecal stasis.

Persistent non-filling associated with some or all of the above additional signs would suggest that inflammatory swelling or adhesions from chronic inflammation had occluded the lumen or caused fibrosis of the musculature, preventing the normal physiological function.

The question of previous operation must always be raised where there is persistent non-filling, for adhesions and tenderness on palpation may, and often do, exist after appendicectomy. Mistakes in this regard are more than possible when the radiologist is asked to express an opinion upon a film alone and practically no history is given.

Thus it will be seen that if the appendix is not definitely outlined at the first or subsequent examination within a few hours later, it must be regarded as a suggestive sign of pathological change, but is conclusive where definite caecal tenderness, spasm, fixation *et cetera* are superadded.

The following remarks refer to the appendix which does fill and therefore becomes visible and is palpable under the fluorescent screen. If the appendix is visible, is regular in outline, and the caecum and appendix are freely movable and, above all, not tender on direct palpation, then there is every reason to presume that there is no associated pathological change. The normal appendix also completely empties as the meal passes onwards from the caecum.

The length varies considerably, and it is usually the long appendix which takes a considerable time to expel the barium meal after the caecum has emptied. Indeed, barium remnants have remained

for days even after severe purgation. The long appendix, for this reason, should be regarded with suspicion, but only if it remains filled or partly filled, for it is reasonable to regard a degree of stagnation of bowel contents as a predisposing cause of inflammation or as a loss of the physiological power of contraction due to fibrosis from preexisting chronic inflammation.

The normal shape is usually comma or S-shaped, but true or apparent kinks often occur. Apparent kinks are merely a foreshortening of the shadow of a normal curve, and the normal curve is demonstrated on rotation of the patient into another plane. True kinks may be due to a short mesentery or to adhesions, and if constant despite palpation and if associated with local tenderness, they are a suggestive sign of pathological change. As before stated, the appendix may be anywhere in the abdominal cavity or peritoneal sacs associated therewith, but the usual position is in the right iliac fossa, below and medial to the caecum.

Variations from the normal position are either congenital or acquired, the latter usually resulting from adhesions drawing the caecum out of its usual position and carrying with it the appendix. Slight variations from the normal position which are considered to be associated with chronic inflammation are the retrocaecal and the right lateral positions. The former is best demonstrated when the caecum is partly empty, otherwise the dense barium shadow of the full caecum obscures the position of the appendix behind it, and it is often difficult to distinguish the appendix posteriorly, even when the patient is rotated into a lateral position.

The normal appendix should be freely movable and not tender on palpation. A short mesentery will restrict free movement, but in the absence of definite tenderness or other sign this restricted movement is of little significance. If it is fixed in position due to adhesions, efforts to separate its shadow from the caecum or adjacent viscera will fail and the adjacent portions of caecum or colon will move as a whole with it, and the movement will be accompanied by local tenderness.

Additional signs will be an irregular outline and possibly constant kinks. Adhesions to the tip of the appendix only may be present. Here the appendix is movable, with the exception of the tip, and during palpation the appendiceal shadow may elongate and return to its former position. Constant strictures of its lumen may also be noted. Adhesions in the caecal region are manifested by fixation, irregularity in outline, ileo-caecal stasis, and associated pain on palpation.

Fæcoliths may be found associated with constant irregularity of the lumen. These may be calcified and in a plain skiagram may simulate a right ureteric calculus. If the appendix is outlined sufficiently well, these concretions appear as rounded or ovoid areas of less opaque substance, usually towards the tip of an appendix with an irregular lumen. Foreign bodies may sometimes be detected.

One case I recall was that of a patient who was suspected of having a chronic appendicitis; when he was examined it was discovered that several small shot had lodged in the appendix and had presumably been there for some considerable time, as he had not eaten game for some months. They were present at operation in a chronically inflamed and fibrosed appendix.

Incomplete evacuation of barium residues from its lumen some hours or even days after the caecum and bowel have emptied indicates a definite chronic fibrosis of its walls and is not necessarily associated with the long appendix, as above mentioned.

Caecal stasis and ileo-caecal or ileal stasis may be a further sign. The normal evacuation time for various types of colon varies considerably, but as a rule the colonic content moves evenly onwards to the distal part of the hepatic flexure. True caecal stasis is shown when the caecum remains partly filled and the remainder of the colon, except the sigmoid and rectum, is practically empty.

Portion of the opaque meal may still be present in the terminal ileum at twenty-four hours or longer after the ingestion of the meal, and this usually indicates the presence of adhesions in the region of the ileo-caecal valve. The thin wisps of meal in the terminal part of the ileum can be mistaken for the appendix unless careful examination is made and technically good X ray films are obtained. Ileal stasis alone only suggests pathological change, for there are other causes than chronic appendicitis. The same applies to caecal and colonic spasm.

A further sign which has been quoted in the past as suggestive of pathological change is insufficiency of the ileo-caecal valve observed in colonic examinations by the barium enema. This phenomenon is of no pathological significance. In fact it is the degree of filling which the radiologist tries by palpation to obtain in all enema examinations of the colon, for he then knows that filling has been complete. Even with such filling it is a rarity to outline the appendix, which is a cul-de-sac. Insufficiency of the ileo-caecal valve is seen in cases in which the appendix has been previously removed, and also in cases in which there is no clinical sign or suggestion of chronic appendicitis.

I have purposely left the sign of pain on direct pressure over the caecum or appendix as the last consideration, for it may be quite definitely stated that most of the other signs lose a great deal of their conclusiveness without the accompanying sign of pressure pain on palpation. This sign is never absent when several of the other signs previously mentioned definitely exist, but it is the most significant one when the other signs are suggested but not conclusively proven to be present.

It may be quite true that "few normal appendices have been found by surgeons and pathologists", as above mentioned, but from my previous remarks it will be seen that a little more than "any grounds whatever" is required for a correct diagnosis of chronic appendicitis, especially when the radiologist is called upon to assist in the elucidation of vague abdominal symptoms which clinically may only sug-

gest appendicitis and which might quite easily be due to chronic cholecystitis, duodenal ulcer or visceroptosis *et cetera*.

Recently I carried out a Graham's test of the gall-bladder in a girl of sixteen years who had had a "chronic appendix" removed some twelve months previously. Her gall-bladder contained a number of transradiant calculi.

Cholecystitis and appendicitis resemble one another clinically, and the fact that cholecystitis is a disease of early life, with later manifestations, is often forgotten.

Finally, an appendix which fills and empties satisfactorily and which is freely movable and not tender on direct palpation, must be considered to be normal, even though caecal spasm, irregular lumen, and other minor signs mentioned above, may also be present at the time of examination.

Reference.

(1) R. D. Carman and A. Miller: "Diseases of the Alimentary Canal", 1917, page 517.

A CONTRIBUTION TO THE STUDY OF THE HEREDITY OF BLOOD GROUPS IN AUSTRALIAN FAMILIES.

By EVA A. SHIPTON, B.Sc., M.B. (Sydney),
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OWING to the importance of the theory of the heredity of blood groups in medico-legal work, some figures are here presented for the population of Sydney.

Material for this work was obtained mostly by the examination of families consisting of father, mother and child, through Saint Margaret's Hospital. A few were obtained from adult families either in private or hospital practice, as opportunity offered.

Nomenclature.

The classification of the blood groups according to Jansky has the right of priority, and in 1921 *The Journal of the American Medical Association*[1] adopted this classification and recommended it for universal use. However, many institutions and countries continued to use the classification according to Moss, and this has been the source of endless confusion. In order to remedy this, the properly qualified international authority, namely, the Commission for the Standardization of Sera of the Public Health Committee of the League of Nations, has entirely done away with numerals and has adopted an alphabetical terminology corresponding to the agglutinogen content of each group, that is, O, A, B, A B.[2][3] In 1927[4] the American Association of Immunologists adopted this classification, which has now been widely accepted throughout the world. Its utility is obvious, as all sources of confusion which may be exceedingly dangerous are eliminated, it has a more scientific basis, teaching is simplified, and, most important of all, it facilitates communication between workers in different countries.[5]

This terminology should now be adopted by all hospitals, blood donors' associations and medical schools throughout this country and the old terminologies of Moss and Jansky should be discarded.

The following shows the relation between the old and new terminologies:

Group O corresponds to Jansky I and Moss IV. No agglutinogens are present in the cells, but both agglutinins anti-A and anti-B are present in the serum.

Group A corresponds to Jansky II and Moss II. The cells of this group contain the agglutinogen A, and the serum the anti-B agglutinin.

Group B corresponds to Jansky III and Moss III. The cells of this group contain the agglutinogen B, and the serum the anti-A agglutinin.

Group A B corresponds to Jansky IV and Moss I. The cells of this group contain both agglutinogens, but the sera does not contain either of the agglutinins.

The question of sub-groups containing additional agglutinogens and agglutinins, as described by Guthrie and Huck[6] and Simson[7] and other workers, will not be discussed here. Suffice it to say that in all the blood of adults in this series of examinations no expected reaction failed to occur and no unexpected reaction took place between specimens of blood of the same group. It still remains to be proved whether the sub-groups described are true sub-groups or whether the reactions are due to quantitative differences.

The agglutinogen is contained in the stroma of the red cells,[8] and Halleur[9] has shown that it is soluble in water and that extracts of the stromata, and also water in which erythrocytes have been washed, contain the agglutinogen. With some erythrocytes the agglutinogen was more soluble than with others. The chemical composition of the agglutinogen is complex.[10][11] These individual characters of the blood, where they are easily demonstrable, have been shown to be only a special example of properties which all the cells of the body possess, and the presence of these group-specific antigens has been demonstrated in various kinds of fixed somatic cells.[12]

The isoagglutinins present in the blood serum can be demonstrated in the breast milk of nursing women,[13] in pathological fluids as transudates and exudates (example, Family 9).

According to Rona and Krebs[14] and Bleyer,[15] isoagglutinins are linked with the euglobulin fraction of the serum and only traces are present in the pseudoglobulin fraction.

Toda[16] has demonstrated that the platelets of human blood are not able to be relegated to the four groups by means of typing sera. Some work by Doan[17] suggests that leucocytes possess individuality and that there is a definite incompatibility between the blood plasma of some individuals and the white blood cells of others.

Constancy of the Blood Group in an Individual.

The blood group of an individual is an immutable constitutional characteristic. It remains unchanged throughout life and can be altered neither by disease, medicaments[18] nor blood transfusions.[19] Variations may occur, and have frequently been reported, in the strength of the agglutinogens and titres of the agglutinins. Harper and Byron[20] have noted the effect of diet on isohæmagglutinins, especially amongst individuals belonging to Group B.

Edgecombe[21] tested the blood serum of eighteen women at various periods of pregnancy and after delivery and found that the fœtus had a definite influence on the titre of the mother blood, most marked with a fœtus of a group different from the mother, where the rise in agglutinating titre of the cells averaged 83%, and of the serum 330%.

Hübener[22] reports an instance in which the expected agglutinogen A could be demonstrated only after recovery from illness. These variations in titre of serum and variations in the agglutinability of the cells, together with pseudo-agglutination, which is especially apt to occur in disease, account for the erroneous conclusions drawn by a few workers.

The work of Huck and Peyton[23] demonstrated that there is no change of blood groups after ether anæsthesia, which was claimed by Levine and Segall.[24] Thomsen and Kettel[25] have obtained results on a large amount of material and find that agglutinins vary in their titre at different ages, and in old age the titre gradually lessens until it can fall as low as in infancy, a process of serological involution. The agglutinogens develop early in fœtal life and have been described in the blood of fœtuses of four, five and six months;[26][27][28] Jones[29] demonstrated them in a seven months fœtus. Karshner's findings[30] confirm the observations of de Biasi[31] that the corpuscles of new-born infants have their full quota of receptors (agglutinogens) and that the distribution of blood groups in infants is the same as in adults.

A few cases are on record in which delay in the maturation of the agglutinogen has occurred.[32] Smith[13] reports two cases in which an O group mother had passed on her agglutinins to her new-born infants and the cells of the umbilical blood were inagglutinable. In a few days, however, agglutinogens were present in the blood of the infants, who belonged to Groups A and B.

These two cases are of great importance and show that umbilical blood should never be used for forensic examinations. According to Thomsen and Kettel, agglutinogens also undergo a process of maturation and the titre rises slowly up to the age of fifteen to twenty years.

The agglutinins are much later in developing than the agglutinogens. Happ showed that at birth and during the first month of life isoagglutination is rarely present; but the percentage of infants in whom the isoagglutinin group is established increased with age, so that after one year the group is usually established, and after two years is always present, as in adults. Jones,[29] by using special methods, was able to demonstrate agglutinins in a much larger percentage of cases than Happ, and concluded that 100% of new-born infants can be correctly grouped by means of their cells.

The appearance of the agglutinins in infants can be hastened by certain stimuli, as blood transfusion.[33]

Sussman[34] states that the new-born child inherits group-specific agglutinogens, but not isoagglutinins, and that the inhalation of particles of foreign saliva suspended in the air gives rise to an immunological formation of isoagglutinins directed against the agglutinogens which the child does not possess. This process is gradual and presumably must vary with the population of the district.

In the series of infants all between seven and nine days old, which form the large part of the material for this paper, it was the exception rather than the rule to be able to demonstrate agglutinins by the ordinary method.

Heredity of the Blood Groups.

In this discussion it is presumed that the reader possesses a knowledge of the Mendelian theory of heredity.

In 1910 von Dungern and Hirszfeld published the first systematic work on the inheritance of the blood groups and brought forward their theory of inheritance in agreement with orthodox Mendelian principles regarding A and B as dominants. According to this theory, the blood group of an individual is the result of four characters united in two allelomorphic pairs, A and not-A, B and not-B, completely independent of each other and situated far apart in the chromosome system. The four blood groups can be formed by the combination of the two pairs of allelomorphs, each pair giving rise to three genotypes and two phenotypes. For example, for the pair A and not-A the following would hold:

A A, dominant homozygote
A, not-A, dominant heterozygote } Phenotype A.
Not-A, Not-A = recessive homozygote.

And the same for B.

The combination of the two pairs therefore gives rise to nine genotypes and four phenotypes.

TABLE I.
Blood Groups and Hereditary Types (after Hirszfeld).[35][36][37]

Phenotypical Groups.	Genotypes.			
O ab	Not-A, Not-A. Not-B, Not-B pure.			
A b	A A. Not-B, not-B pure.	A, not-A. Not-B, not-B hybrid.		
B a	Not-A, not-A. B B pure.	Not-A, not-A. B, not-B hybrid.		
AB c	A A. B B pure.	A, not-A. B, not-B di-hybrid.	A, not-A. B B mono-hybrid.	A A. B not-B mono-hybrid.

In 1924 the mathematician Bernstein pointed out that this theory was not compatible with deductions concerning random mating in mixed populations. Bernstein[10] claims that the hypothesis of three allelomorphic genes, A, B and R (O), one of which is present in each member of a certain pair of chromosomes, A and B being dominant over R (O), agrees more closely with observed facts. Since each somatic cell possesses two of these gametes—one from the father and one from the mother—the following genotypes and phenotypes occur (Table II).

TABLE II.

Genotype.	Phenotype.
RR (OO)	O
AA, AR	A
BB, BR	B
AB	AB

On the basis of elaborate analyses of available data by geneticists Bernstein's hypothesis may now be regarded as a satisfactory account of the inheritance of the blood groups.

In comparing the two theories it is to be noted that there is no disagreement about the facts most essential for the practical application of the tests, namely, "the biochemical structures of A and B never make their appearance in the offspring unless they are present in one or both of the parents".

According to the theory of von Dungern and Hirszfeld, an O parent can have a child A B and a parent A B can have a child O; according to Bernstein's theory this is impossible.

Bernstein's theory regarding this particular point is supported by an impressively large number of observations, the exceptions occurring mostly in the older literature, where the possibility of error was great. However, excluding any doubtful reports, there is one instance reported[38] in which a mother of group A B gave birth to a child whose blood was indistinguishable from Group O. According to Landsteiner,[39] it is therefore wisest to adopt a conservative attitude regarding the possibility of parent-child combination A B-O or O-A B. In this series it is interesting to note that the offspring in five families with one Group A B parent are all either Group A or Group B.

Further reliable work on a suitable section of the population is required finally to settle the matter.

Technique and Methods.

All the bloods were collected, labelled and examined personally. Blood was collected from the fathers when they visited their wives in the evenings, and from the mothers and babies between the sixth and ninth days of the puerperium. In no case was blood taken if either of the parents made any objection, no matter on what grounds. In the adults a few drops of blood were collected from a prick on the finger, some into 3·8% citrate solution and some into a clean, dry tube, in order to collect the serum. With the babies, blood was taken from a prick on the heel. It was not always possible to collect the blood in the post-digestive period. After collection the blood was placed on ice for twenty-four to forty-eight hours. The cells were washed once with citrate solution. This was done for two reasons: (i) the supernatant citrate solution from the babies' cells was frequently stained with bile pigment, (ii) to eliminate the possibility of the red corpuscles retaining sufficient of their own serum to interfere with the reaction.[40] The actual agglutination tests were performed on plain, clean glass slides, equal parts of serum and cell suspension being used. The cell suspension was always between 2·5% and 5·0%. The slide was oscillated and final readings were not taken until the end of fifteen minutes, but agglutination, when present, usually occurred within two or three minutes. It was noted that the A-a system agglutinated more quickly and strongly than the B-b system.

The sources of error tabulated by Ottenberg were carefully avoided.[41] As can be seen from the results, the inability to demonstrate agglutinins in babies' serum was a frequent occurrence. Whether these are entirely absent or only not demonstrable by this method is another question. It is known that agglutinins can be strengthened by alternately freezing and thawing the serum over a period of weeks, when the serum separates out into two layers, the upper colourless, like water, and not containing any agglutinin, the lower straw-coloured and containing the strengthened agglutinin. Unfortunately facilities for refrigeration were not available when this work was being done and this method could not be tried, so has had to be postponed until later, when I hope to be able to obtain another series of families and test the serum from the babies in this manner.

The agglutinogens, when present in the babies' blood, were easily demonstrated. In the cases in which neither agglutinogens nor agglutinins were present, the baby was classified as belonging to Group O.

Results of Investigation.

Altogether 81 families with 91 children, comprising 253 individuals, were examined. Of these individuals, 119 belonged to Group O, 107 belonged to Group A, 21 belonged to Group B, and 6 only belonged to Group A B.

There are no exceptions to Bernstein's law, as can be seen from Table III and results in detail of each family (Table IV).

Although the father's blood could not be obtained in the last case shown in Table IV, it is included because of the importance of the question of A B mothers.

These results agree with those obtained by previous workers in this field and are of the utmost importance from the medico-legal point of view.

No attempt has been made to review the literature, which has been adequately done by others; but some points bearing on the subject have been shortly discussed.

TABLE III.

Parental Combination.	Number of Families.	Number of Children in each Group.			
		O	A	B	AB
O+O	15	18			
A+A	17	5	12		
O+A	32	21	18		
B+B					
O+B	8	4		4	
A+B	4			3	1
O+AB	3		1	2	
A+AB	2		2		
B+AB					
AB+AB					
Total	81	48	33	9	1

TABLE IV.

Number of Family and Initials.	Father's Group.	Mother's Group.	Child's Group.	Remarks.
(1) S	O	A	(1) O (2) O (3) A	Adults.
(2) S	O	O	O	No agglutinins or agglutinogen in babies' blood.
(3) P	O	A	O	Baby first examined at 21 days. Reexamined at 37 days. No agglutinins or agglutinogens.
(4) B	O	A	A	Adult.
(5) O	O	A	O	No agglutinins or agglutinogens.
(6) G	B	O	O	Both agglutinins in baby's serum.
(7) B	B	O	O	Agglutinins present, but feeble.
(8) P	A	O	A	No agglutin in serum.
(9) S	A	A	(1) O (2) O	Adults. Mother had ascites, due to cirrhosis of liver, with an anæmia of the hyperchromic macrocytic type and the ascitic fluid agglutinated B cells.
(10) A	A	O	O	Both agglutinins present in baby's serum.
(11) R	O	A	O	Mother's agglutinin anti-B very weak. None demonstrable in baby's serum.
(12) S	A	O	O	No agglutinins in baby's serum.
(13) I	O	A	A	No agglutinins in baby's serum.
(14) R	O	A	O	No agglutinins in baby's serum.
(15) B	A	A	A	No agglutinins in serum.
(16) R	O	O	O	Anti-A agglutinin strong, B weak.
(17) C	A	O	O	No agglutinins.
(18) P	A	O	A	No agglutinins.
(19) V	O	O	O	No agglutinins.
(20) J	A	A	A	No agglutinin.
(21) R	A	O	O	Both agglutinins present.
(22) F	O	A	A	No agglutinin.
(23) V	O	A	O	No agglutinins.
(24) S	A	A	A	No agglutinin.
(25) C	O	B	O	No agglutinins.
(26)	A	B	B	Mother in (25) C.
(27) C	A	A	A	No agglutinin.
(28) K	A	O	O	No agglutinins.
(29) B	A	A	O	No agglutinins.
(30) S	A	A	A	No agglutinin.
(31) S	A	A	A	No agglutinin.
(32) H	A	B	B	No agglutinin.
(33) R	O	A	Child nine days O Child two years A	Both agglutinins. Agglutin anti-B present.
(34) L	O	A	O	No agglutinins.
(35) R	O	A	A	No agglutinins.
(36) P	O	O	O	Children, adults, three sons and a daughter.
(37)	O	A	O	Child adult.
(38) P	B	O	B	Weak agglutinin anti-A.
(39) C	AB	O	B	Strong anti-A agglutinin.
(40) M	O	A	A	Weak agglutinin anti-B.
(41)	A	A	O	No agglutinins.
(42) K	O	O	O	No agglutinins.
(43) M	B	O	O	No agglutinins.
(44) M	A	O	A	No agglutinins.
(45) A	A	A	A	No agglutinins.
(46) J	O	O	O	Weak agglutinins.
(47) C	O	B	B	Weak agglutinins.
(48) H	O	A	A	No agglutinin.
(49) O	A	O	A	No agglutinin.
(50) T	O	O	O	No agglutinins.
(51) E	A	O	Twins O A	No agglutinins. No agglutinins.
(52) B	O	A	A	No agglutinins.
(53) G	O	AB	B	Agglutinin present.
(54) W	A	O	Twins O A	No agglutinins. No agglutinins.
(55) L	A	A	Adult A	Agglutinins present.
(56) Q	AB	O	A	No agglutinin.
(57) O	A	A	A	No agglutinin.
(58) M	AB	A	A	No agglutinin.
(59) O	O	A	O	No agglutinins.
(60) L	A	AB	Adult A	Agglutinin present.
(61) G	O	O	O	Agglutinins present.
(62) R	A	A	A	No agglutinins.
(63) C	A	A	A	No agglutinin.
(64) M	B	O	B	Agglutinin present.
(65) B	O	O	O	No agglutinins.
(66) M	O	O	O	No agglutinins.
(67) E	A	O	A	No agglutinin.
(68) V	O	A	A	No agglutinins (erythroblastosis).
(69) B	B	O	B	No agglutinin.
(70) B	A	O	O	Both agglutinins present, strong.
(71) H	A	A	A	No agglutinin.
(72) B	A	O	A	No agglutinin.
(73)	A	B	AB	No agglutinins in serum.
(74) D	O	O	O	No agglutinins.
(75) G	A	A	O	No agglutinins.
(76) B	O	O	O	No agglutinins.
(77) L	A	A	A	No agglutinin.
(78) O	O	O	O	Agglutinin anti-A present, but no anti-B.
(79) H	O	O	O	No agglutinins.
(80) E	A	B	B	No agglutinins.
(81) W	O	A	Two children, O and A, 4 and 6.	All agglutinins present.
(82) C	?	AB	A	No agglutinin.

It is necessary to emphasize the necessity for care in technique and the necessity for the worker to possess an adequate knowledge of all sources of error *et cetera* which may arise in the work, especially when dealing with medico-legal cases, as an error in grouping may have serious consequences. If any deviations from Bernstein's law occur during the course of an investigation, all possible sources of error must be examined.

Acknowledgements.

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AUTOHÆMOTHERAPY IN THE TREATMENT OF BRONCHIAL ASTHMA.

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ENCOURAGED by the alleviation afforded to sufferers from chronic eczema attending the Dermatological Department of the Royal Prince Alfred Hospital following the empirical use of injections of the patients' own blood, we desired to test for ourselves the exact value of this procedure in adults and children suffering from bronchial asthma. The patients selected were all under the age of forty-five years and suffered from typical paroxysms. Twenty-four were children under the age of sixteen years, seventeen were adults all of whom, except two, were below forty years of age, and two others afforded typical examples of vasomotor rhinitis and were about forty years old.

The largest number of patients so treated that we can discover from the literature is sixteen. Henske, the author, stated that he was satisfied with the improvement in nine of these persons. Each of our patients was first asked to describe the frequency and severity of his attacks and, where possible, independent corroboration of his history was obtained. He then underwent a careful physical examination in which all other possible causes of dyspnœa were excluded. A special examination of his ears, nose and throat was made to exclude the presence of polypi or focal infection, a radiogram was taken of the lung fields, and the patient's skin was tested by the scratch method for the common allergens. In the children the paranasal sinuses were examined radiographically and a fractional gastric analysis was performed. All advice and drugs which might favourably affect the incidence of the disorder were deliberately withheld, and if, as was usual, the patient was already taking one of the common mixtures prescribed for asthma, he was allowed to continue doing so. Ten cubic centimetres of blood were then removed from the median basilic vein and injected forthwith into the buttock without admixture of citrate or any attempt to separate the blood elements. This was repeated five times at intervals of a week. The patient then entered upon a period of observation varying from three to twelve months. The patients presented themselves at intervals to report and for observation or, in a few instances, communicated monthly with us by letter. They were particularly requested to compare the frequency and character of the attacks with those experienced before treatment, and were deliberately influenced, as far as possible, to doubt or belittle the value of the treatment.

The question of assessing the results obtained by any form of therapy in allergic disease is rendered extraordinarily difficult by the constant flux in the allergic state and by the many factors, psychological, physiological and climatic, which can potentially influence it still further. It is also difficult to understand how such a procedure as autohæmotherapy can operate other than wholly psychologically. Whether benefit may be derived from the release of peptone from the tissue digestion of the blood in the gluteal muscles or from the injection of minute amounts of Oriel's proteose (if this is still to be considered of importance), or from some other element of the blood, it is impossible for us to say.

We would therefore prefer merely to record our own experience of this method as an empirical experiment. In some instances we were able to substantiate the patient's own statements as to benefits received by questioning relatives and friends and by our own observation of an increase in weight or other obvious improvement in general health.

Results.

It was soon obvious that, as would be expected, any beneficial results obtained were directly proportional to the youth of the patient, and therefore to the less complicated character of the asthma. In general it may be said that the purer the allergy, the better the results obtained from the injections. It was therefore necessary to divide the series of 43 patients, of whom 25 were male and 18 female, into two classes.

Class I included patients in whom, as far as we could discover, the injections had a fair and unhindered opportunity of affecting the course of the disorder: 25 patients.

Class II included patients in whom other factors, such as a change in place of abode or other conditions of domestic environment, or the existence of nasal or paranasal sepsis, or abnormalities in the anatomy of the chest, might possibly have had an independent effect on the reading of the results. Included here also are those instances in which previous remissions for as long as the period of observation have occurred, either spontaneously or following tonsillectomy or other methods of treatment: 18 patients.

The results obtained in each class are described in terms of groups as follows:

Group I: No attacks of significance during at least six months.

Group II: Much fewer attacks, of lessened severity.

Group III: Attacks as frequent during period of observation, but less severe.

Group IV: No improvement whatever.

The results are set out in Table I.

TABLE I.

Patients.	Group I.	Group II.	Group III.	Group IV.	Total.
Class I	8	10	5	2	25
Class II	5	4	5	4	18
Total	13	14	10	6	43

A similar subdivision of the 24 children is set out in Table II.

TABLE II.

Patients.	Group I.	Group II.	Group III.	Group IV.	Total.
Class I	6	4	4	0	14
Class II	3	4	3	0	10
Total	9	8	7	0	24

It is at once apparent that the young asthmatics constitute by far the greater part of those patients showing improvement, and in approximately three-quarters of the children the attack frequency was appreciably reduced, while in every instance some alleviation in severity was manifest. Incidentally, in the children, it was observed that hæmotherapy was more successful with children of the first decade than with those of the second; that the presence of skin sensitivity to the scratch method presaged a better result from autohæmotherapy; and that the average age of onset of asthma in this series was no earlier in children with a family history of the disease.

Discussion.

As stated above, we are conscious of the many fallacies to which we are liable in assessing the results in a small series of patients suffering from such a capricious disease. We shall therefore refrain from any dogmatic conclusions, beyond stating that we are left with a distinctly favourable clinical impression of the value of this simple procedure. Thankful expressions of the patients themselves, their wish in some instances to repeat the course, and the obvious improvement in the colour and nutrition, particularly of the children, consequent upon the palliation of their asthma, have been sufficient reward for the conduct of this work in the busy out-patient clinic. We feel that the improvement, especially in the adults, is short-lived; but it may serve to give them confidence and sleep, which are so extremely important in stiffening the mechanism of the asthmatic machine gun.

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Reports of Cases.

THROMBOSIS OF THE COLIC VESSELS.

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ON November 6, 1933, D.B., a male, aged fifty-five years, following the occupation of a butcher, was admitted to the Royal South Sydney Hospital under my care with the following history.

He had complained of the presence of double inguinal hernia for the past four years, which had given no trouble until three days previously, when the hernia on the left side became tense, irreducible and painful. He had had no vomiting and the bowels had acted during this time. The hernia was apparently reduced by the house surgeon. On the following afternoon his abdomen was somewhat distended and he appeared rather collapsed. A small enema was given, which resulted in pain, further collapse and retention of the enema. A rectal tube was passed and the bowel was emptied. In view of this the possibility of a reduction *en masse* or of a Richter's hernia was considered and operation was decided upon.

At operation an old hernial sac was discovered, containing some adherent pelvic colon and omentum. There were insufficient findings in the sac to account for the patient's collapsed condition. After freeing adherent bowel and omentum and returning them to the abdomen, a small quantity of pus was seen to escape from the upper part of the wound, causing no little surprise, in view of the absence of rigidity and the slight nature of the tenderness previously observed. Extension of the wound in an upward direction revealed gangrenous bowel, and the incision was extended to the costal margin. It was now seen that about 3·75 centimetres (one and a half inches) of the transverse colon, the splenic flexure, the descending colon and the iliac colon were gangrenous, with an area of about 5·0 centimetres (two inches) in diameter just above the junction of the descending and iliac portions of the colon, on the antemesenteric border, showing the most advanced degree of damage. The gangrenous area was rapidly and widely resected. The free and healthy ends of the transverse and of the pelvic colon were sutured in approximation and brought out of the wound as a shotgun colostomy. The wound was closed with drainage of the posterior pouch and the patient was returned to the ward in fair condition.

Considering the gravity of the lesion, the patient's condition was remarkably good following the operation. The colostomy commenced to work within twenty-four hours and at no time gave trouble in this direction. The wound, as was to be expected, broke down and suppurated. The temperature did not rise above 37·8° C. (100° F.) and the pulse rate fell from 152 to about 100 by the fifth day. There was little inclination for food, but fluids were taken well. On the seventeenth day there was a rise of temperature to 38·3° C. (101° F.), and the following day some pus was evacuated from the loin and the temperature again subsided, improvement recommencing and progressing steadily. This rise in temperature was accompanied by a slight cough, which very soon cleared, and by the twenty-third day the patient was comfortable, taking his food well, showing an interest in life, and his wound was healing satisfactorily. His colostomy was working well, although portion of the upper loop had sloughed, and he did not have a great deal of this loop showing. The progress was maintained until the thirty-seventh day, when his temperature again rose to 38·9° C. (102° F.), accompanied by a rise in pulse rate. This had subsided within three days and he was again fit and well and was out of bed on a lounge in the sun.

On the forty-fifth day his temperature again rose to 37·8° C. (100° F.), and three days later had risen to 39·8° C. (103·8° F.), accompanied by coughing and a slight rise in the respiration rate and sputum. The sputum increased in quantity and became offensive, his temperature was remittant and remained up until the fifty-fourth day, when death occurred.

An X ray examination of his chest, made on December 27, revealed an unresolved pneumonia with a centrally placed cavity in the upper left lobe. The question of bronchoscopic aspiration was considered, but the patient was not thought well enough to undergo this procedure.

Unfortunately permission for a *post mortem* examination was not obtained.

The examination of the bowel removed at operation revealed the following. Macroscopically the left colic artery contained a recent thrombus, while the veins were patent. The bowel, including the left 5·0 centimetres (two inches) of the transverse colon, the descending and the iliac colon, was a dark purple colour, with the changes most marked just above the junction of the descending and the iliac colon, where there was a patch on the antemesenteric border, 5·0 by 3·75 centimetres (two by one and a half inches), where the gangrenous mucous membrane was bulging through the thinned muscular coats of the bowel. At this situation perforation was obviously imminent. Opening the bowel showed the mucosa of a greenish colour and apparently separated from the muscular coating. The adjacent transverse and pelvic colons were thickened and œdematous.

Microscopically, sections of the affected part of the bowel showed acute inflammatory changes with numerous collections of neutrophile cells in the mucous membrane and muscular coats. Some necrotic areas were present and many of the blood vessels were engorged with blood cells.

It would appear, in view of the subsequent history of the patient and the fact that the rest of the abdomen was normal at operation, that the condition was of an embolic nature due to vegetation on the heart valves. This, however, is purely surmise.

Discussion.

Of the reported cases, such as I have been able to trace, four in number, three were in women varying between the ages of twenty-seven and sixty-four years, and one in a man aged fifty-five years. Two were mentioned by Lockhart-Mummery, who quotes Littlewood, of Leeds.

Littlewood's patient, a woman of sixty-four, had a history with several days of complete intestinal obstruction, with fæcal vomiting, abdominal distension and without any history of melæna. Operation revealed a portion 6·75 to 7·5 centimetres (two and a half to three inches) of the left half of the transverse colon thickened and œdematous, with the bowel wall markedly injected. The corresponding portion of the mesocolon was similarly thickened. The mucous membrane in this area was a chocolate colour and slightly swollen and sharply demarcated from the normal mucosa. A thrombus was found in a small artery near the bowel, the clot not being attached to the vessel wall. The bowel above the affected area was distended back to the caecum, while distal to the obstruction the bowel was collapsed. The remainder of the abdominal organs were normal. This patient died on the operating table.

Again, Lockhart-Mummery quotes T. H. Gillet in reporting a second case of a woman, aged forty-nine years, seized with a sudden abdominal pain followed by blood-stained diarrhœa within forty-eight hours. Nausea and severe abdominal pain were the symptoms on the third day. The abdomen was opened and the transverse colon was found to be a dark purple in colour and much thickened. The ascending and descending portions of the colon were anastomosed. The patient died two days later, apparently from toxæmia. There were numerous small hæmorrhages into the bowel, but no clots could be discovered in the branches of the middle colic artery. There was an old mitral lesion of the heart with some vegetations.

Cokkinis reports a case of a woman, aged twenty-seven years, with a history of sudden onset of acute pain in the right side of the abdomen, accompanied by vomiting. On the fifth day the clinical picture suggested an appendiceal abscess. The abdomen was opened. The appendix was found healthy, but a gangrenous area of 15·0 centimetres (six inches) of the transverse colon, with gangrenous omentum attached, was found. The gangrenous mass was resected and a lateral ilio-sigmoidostomy was performed. Six days later a fæcal fistula developed, and on the eleventh day there were signs of consolidation at the bases of both lungs, with a pleural rub on the right side. The patient became rapidly worse and died five days later. The histological report showed acute phlegmonous inflammation with ulceration necroses. *Post mortem* examination showed that the ileo-colostomy had broken down, with the formation of a fæcal fistula. The caecal mucosa was completely gangrenous. There was an organized thrombus in the right colic artery. There was a double pyosalpinx with pus present in the pouch of Douglas. The lung showed a purulent bronchopneumonia, with areas of gangrene in the upper lobe of the left lung. A thrombus was found in the pulmonary artery, at the apex of the gangrenous portion of the lung.

A second case reported by Cokkinis was that of a man, aged fifty-five years, who had been in bed for eight weeks with bronchopneumonia. Seven days before being seen he had a sudden and severe abdominal pain, which had persisted and which was accompanied by continuous vomiting. The bowels had acted normally. The lower part of his abdomen was rigid and distended. Death occurred on the following day. A *post mortem* examination revealed purple hæmorrhage with moist swelling of the wall of the caecum, the first ten centimetres of the colon and the last six centimetres of the ileum. The next fifteen centimetres of the colon were in a similar condition of infarction. There was partial necrosis and local hæmorrhagic swelling of the small intestine, except in its upper 210 centimetres (seven feet). The mesentery of the lower half of the ileum was shrunken and partially necrosed. There was a lightly adherent friable embolus 0·4 centimetre in diameter blocking the common origin of the ileo-colic and right colic arteries. The aorta and other arteries were atheromatous. The lung showed an organized bronchopneumonia.

Ætiology.

From these cases it is seen that there is no constant ætiological factor. Sex does not seem to play a part, nor is age a material point. Associated pathological conditions vary. In one case there were vegetations on the heart valves, in another atheroma, while in a third a pyosalpinx is suggested as the root of the evil. In the case being reported the subsequent late pulmonary embolus makes one suspect cardiac vegetations as the origin of the original thrombus.

Symptomatology.

The first symptoms would appear to be an acute abdominal pain of sudden onset followed by blood-stained diarrhœa and later by signs of intestinal obstruction. Collapse also seems to be of constant occurrence. A rise in temperature would not appear to be constant, but an increase pulse rate is the rule. An accurate diagnosis would not be possible prior to operation; it would only be possible to say that there had been an intraabdominal catastrophe.

Treatment.

Early operative interference seems to give the only prospect of recovery, and wide resection should be the correct procedure. Short-circuiting would not remove the cause of the toxæmia, which in itself would lead to a fatal termination. In view of the underlying ætiological factors, the prospects of recovery are not great, for even should the bowel recover, as in the case under consideration, further complications, in the way of additional embolic processes, pulmonary infarcts *et cetera*, are more than likely to occur and lead to a fatal conclusion.

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Reviews.

STERILITY.

In his work on human sterility Dr. Samuel Raynor Meaker presents a valuable study of its causation, diagnosis and treatment.[1] The book comprises three main sections, dealing with causation of human infertility, diagnostic study of the sterile mating, and treatment of sterility. The analysis is most searching; nothing is left to the imagination. Thus we have a close survey of deficient spermatogenesis, testicular hypoplasia, hostility of prostato-vesicular secretions and allied causes in the male.

In dealing with "female faults" in the causation of sterility, the author stresses "viscosity of the mucus within the cervical canal" and states that "viscid secretions impose a barrier which spermatozoa are rarely able to surmount". Attention is called to what is conceivably "one of the commonest impedances to human fertility", namely, impassivity of the ovario-tubal meatus, the term given by Dr. Meaker to "the open gap between the ovary and the channel by which its externally secreted product is carried off".

In addition to local genital faults (male and female) constitutional factors and endocrinopathy receive full attention. The diagnostic study of sterile mating occupies eleven chapters. For completeness of investigation it would be difficult to find a parallel. A form of instructions with a daily programme of examinations and tests for eight days is issued to patients (husband and wife) who are referred to Dr. Meaker and his colleagues—their names and addresses are given—for these special and general examinations. (It must be noted that this work is published under the ægis of the National Committee on Maternal Health, Inc., New York, of whose board of directors Dr. Meaker is a member.)

Treatment is logically related to the causative factors. Dr. Meaker points out that there are very often more than one in a given case. Due importance is attached to general endocrine treatment. Tubal insufflation with gas and injections of oil, conservative surgery of tubes and ovaries, X ray therapy, are considered. In "uterine blockade" in the hypoplastic uterus Dr. Meaker strongly condemns "surgical dilatation, the insertion of tents, packs or stems and even cutting operations to correct a supposed obstruction caused by anteflexion with angulation at the *os internum*". Yet he favours Reynold's vaginal plastic operation for anteflexion of the cervix and describes his own operation of posterior median discission of the cervix for "poor cervical drainage". The final chapter deals with prevention of sterility and genital hypoplasia, improvement of sex hygiene and control of venereal disease.

Dr. Meaker's book reflects extensive and intensive study. It has a broad philosophical outlook and impresses the reader both by the matter and the manner of presentation. It should appeal alike to the gynæcologist and the general physician.

[1] "Human Sterility: Causation, Diagnosis, and Treatment: A Practical Manual of Clinical Procedure", by S. R. Meaker, M.D.; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 287, with illustrations. Price: 18s. net.

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SYPHILIS THE UNKNOWN.

In opening a discussion on unsettled questions of neurosyphilis at a meeting of the Section of Neurology of the Royal Society of Medicine, S. A. Kinnear Wilson, the President, said that it would be difficult to find another instance in which the research crowned by the detection of the apparently causative organism had started more problems than it had settled, or in which uncertainties still gathered round a question imagined decades ago to have been solved. Speaker after speaker at the discussion brought forward aspects of the subject that seemed to make it still more obscure, and, according to the published account, the meeting closed in much the same obscurity as that in which it was opened. This is not altogether a disadvantage. Problems have to be stated from time to time, lest it be forgotten that they exist; and the medical mind needs to be reminded that unconquered fields are more extensive than those already won. It is true that the causative organism, the *Treponema pallidum* or *Spirochæta pallida*, is known; and also that intensive treatment by certain chemical agents, when undertaken early and with energy, will, as a general rule, result in what is known as a cure. By a cure, of course, we mean that the patient lives for many years without symptoms of the disease, and possibly that his blood serum and his cerebro-spinal fluid do not react to the Wassermann or similar test. Actually we are aware that tests of this kind are not specific, that is, a non-specific antigen may be used in their performance, that they have their limitations, and that these limitations may vary. We also know that the causative organisms in some form or other often lie latent in the tissues of the body, waiting, maybe for years, until something happens to release or to activate them once more. These things we know, but much more is unknown.

Kinnear Wilson, in the Royal Society of Medicine discussion, pointed out that the two names given to the organism were interchangeable and that, since one had a botanical and the other a zoological significance, the nomenclature alone showed how little was known of the organism. He went so far as to say that there were good grounds for questioning the view that it fulfilled Koch's postulates. He then pointed out that astonishingly little is known of the life history of the spirochæte of syphilis. There are good grounds for supposing that it has a complex life cycle, and many workers have upheld this view. Both Meirowski and Grütz described peculiar bud-like formations which they thought were stages in the development of mature spirochætes; but, as Bulloch has pointed out, there is no proof that these supposed involution forms were not artefacts or mere fragments of tissue. E. H. Ross also described appearances that he held were stages in the spirochætal life cycle. Kinnear Wilson, in his paper at the discussion, referred to the work of J. E. R. McDonagh, who described a complex life cycle, from a sporozoite, through sexual forms, to a minute infective spore. Kinnear Wilson said that some of McDonagh's observations had been confirmed. McDonagh, who was present at the meeting, said that the spore of a coccidial protozoon was the actual cause of the infection, the *Spirochæta pallida* being no more than the adult phase in the life cycle. The existence of a spirochætal life cycle should be proved or disproved. If

it could be shown to exist and if its stages were determined, some aspects of the riddle of syphilis could be solved. For example, McDonagh holds that if we accept the existence of a protozoon that has a complicated life cycle which has to be completed before signs and symptoms manifest themselves, we have an explanation of the long incubation period and of conceptional and congenital syphilis; and, further, that we realize why the word cure should never be used in connexion with syphilis. The existence of quiescent forms in a life cycle would also offer a possible explanation for the long periods during which an infection may lie dormant before such conditions as *tabes dorsalis* and general paralysis of the insane make their appearance.

Another of the most important problems in syphilitic infection is the reason for the selection of certain sites for manifestations of the disease. Particularly is this question exemplified in *tabes dorsalis* and general paralysis of the insane. These two conditions were discussed at the Royal Society of Medicine meeting. Reference was made to the views held regarding dermotropic and neurotropic strains of spirochæte. Several workers, including Nichols, Levaditi, Marie and Nonne, have described these two strains. To accept such an explanation appears to simplify somewhat the question as to why the neurones should be picked out in general paralysis and the exogenous nerve fibres and posterior root ganglia in *tabes dorsalis*. If we think on the broadest lines, however, this explanation does not simplify the problem—we have still to determine why syphilis in some persons should attack such an organ as the stomach or the tongue or any other part of the body. This, of course, is a problem in every branch of clinical medicine. Some explanation must be found that will fit in with every type of syphilitic manifestation. Experimental work and clinical investigation have not proved convincing as far as dermotropic and neurotropic forms are concerned; and it has also been pointed out that if a neurotropic strain does exist, it must be to some extent dermotropic, since the neurosyphilitic may transmit his syphilis to another person. Perhaps the most satisfactory hypothesis that has been advanced is that allergy determines the site of a syphilitic lesion. This view has been put forward by different workers in different ways. McIntosh and Fildes thought that the tissues, having become allergic by long interaction between themselves and the virus, responded to an exacerbation of the spirochæte in a manner peculiar to the particular tissue. Gennerich regarded allergy resulting from interaction between the skin and the spirochæte as the important factor—if this was weak by reason of natural non-reactivity of the skin or as a result of treatment, there was correspondingly greater activity of *Spirochæta pallida* in the internal organs. There can be no doubt that allergy plays a large part in the manifestation of many conditions; and it is interesting in passing to note that as a result of some experimental observations it has been held to determine the onset of lobar pneumonia. If allergy be accepted as part of the story of syphilis, other factors will be necessary.

Many other questions remain to be determined, such as the mechanism of the action of malarial therapy in general paralysis, and the reason why some patients respond to such therapy while others do not. They need not be enumerated. To review the present position ought to bring a lively discontent and a desire to further research. Though all medical practitioners cannot undertake research, they can all follow what others are doing. A correspondent to this journal recently objected when it was suggested in these pages that general practitioners should make an intensive study of the treatment of venereal disease. He had fears of the degeneration of the profession. Only if medical practitioners forsake the scientific study of the problems associated with their everyday work will they degenerate; and in such circumstances their degeneration will be as progressive as that found in the spinal cord in *tabes dorsalis*.

MEDICAL BENEVOLENCE IN NEW SOUTH WALES.

THE ninth annual report of the Medical Benevolent Association of New South Wales has been received. On many occasions medical practitioners

have been urged to support the benevolent association in the State in which they reside. It is surprising that urging of this kind should be necessary, and the present report shows the necessity once again. The annual subscription to this association is one guinea; the members of the New South Wales Branch of the British Medical Association number 1,599; and the subscribers to the Medical Benevolent Association last year numbered only 119. In the previous year there were 198 subscribers. The members of the British Medical Association in New South Wales are not very charitably inclined to those of their own profession who have fallen by the wayside.

The chief point of interest in the present report has to do with the acquisition by the Association of the residue of the benevolent fund of the Australian Medical Association. This Association came into being in 1859. It had a benevolent fund limited in scope to its own Fellows or their children. The Association ceased to exist in 1869; and its benevolent fund, amounting to £400, was vested in three trustees. This money gradually accumulated, and in 1934 reached a total of about £3,000. It occurred to Dr. W. H. Crago, one of the trustees, that the funds of the defunct association could best be administered by the Medical Benevolent Association of New South Wales. This, of course, could not be done without the decision of the Equity Court. In spite of the gratuitous advice and help of Messrs. Stephen, Jaques and Stephen, solicitors, of Sydney, the cost of transference amounted to over £405, and this sum had to be taken from the fund. The Medical Benevolent Association of New South Wales is to be congratulated on what may be regarded as a windfall. This windfall should not, however, be used as an excuse for any slackening of interest on the part of medical practitioners. The Honorary Treasurer, Dr. E. S. Littlejohn, has suggested that membership of the Association might be increased if the honorary secretaries of the Local Medical Associations throughout the State were to act as local honorary secretaries to the Medical Benevolent Association and were to use their personal influence in attaining the desired end. This suggestion should be adopted.

Current Comment.

INFECTIOUS ERYTHEMA.

In some of the legends of old there are characters which, for one reason or another, are constrained to remain nameless. In the modern medical world the processes of disease, some of the villains of the drama of public hygiene, are certainly not nameless. The difficulty is to agree as to which name shall be used, for although we must follow the purists in believing that the only correct name is that based on ætiology, in many cases we must be content to coin and use a purely descriptive term. Infectious or, extending the term more widely still, toxic erythema is one of these broad symptomatic names. Excluding the non-infectious varieties, there are many cases of erythema in which the sudden febrile onset suggests an infection, yet either no known group can be found to include the individual case, or intensive investigation cannot be carried out.

Nine years ago an outbreak of epidemic illness occurred in Haverhill, a town in Massachusetts. The patients all suffered a rapid onset of illness, showed a characteristic erythematous eruption, and had involvement of the joints by pain and swelling; the course was apt to be remittent and lasted from days to weeks. Edwin H. Place and Lee E. Sutton have now written a complete report of this epidemic, which was investigated clinically and bacteriologically at the time, and which is of great interest inasmuch as a specific organism was isolated.[1]

A similar epidemic was noted in Pennsylvania in 1925, and it was estimated that 600 cases occurred. Levaditi and others had previously described the isolation of what they termed a streptobacillus from patients who suffered from an acute febrile erythema, and the organism found by Parker and Hudson in the Haverhill epidemic, called by them Haverhillia, is thought to be of the myco-bacterial family. It has recently also been isolated from a few cases in Boston.

The onset was abrupt, with fever, rigors, vomiting and scattered pains in the joints. The fever tended to remit after a few days, and later recurred. There was a macular or papular eruption and sometimes petechiæ appeared. There was swelling and pain in the joints, sometimes with effusion of fluid, and pain and stiffness lasted a variable time after the temperature fell to normal. The resemblance to dengue fever will at once be seen, but the appropriate mosquito vector was not found, and the bacteriological studies demonstrated what appears to be the causal organism in the blood and occasionally in the joint fluid. The origin of the infection was believed to be milk. No direct proof of the presence of the bacterium in the milk was obtained, but all the patients had consumed milk from certain dairies, and after pasteurization the

[1] *Archives of Internal Medicine*, November, 1934.

epidemic ceased. Other indirect evidence also pointed strongly to milk as the source of the illness. As Levaditi and others have shown that rats and mice are natural carriers of an organism which appears to be identical with that isolated during this epidemic, there are interesting possibilities in the preventive field.

The particular interest in this account lies in the local possibilities. Cases of obscure toxic erythema occur from time to time, and these should be carefully investigated. The characters of the rash are of interest, as they may attract the attention of practitioners should similar cases occur. The eruption occurred in the first few days of the illness; it was most intense at the distal ends of the extremities, especially on the extensor and lateral aspects, and often invaded the backs of the hands and dorsal surfaces of the feet. The flexor surfaces were usually free, but the trunk and face were sometimes involved in the severer cases. The rash was irregular and blotchy, dull red in colour, and occasionally petechial. The most troublesome feature of the illness was arthritis. This began early and was always to some extent disabling, and not infrequently prolonged and productive of residual stiffness. This stiffness eventually disappeared, but in some cases lasted for months. The blood showed a slight leucocytosis. No deaths occurred. The tentative diagnosis varied—dengue, influenza, Malta fever (abortus fever) and *erythema multiforme* or *infectiosum* were mentioned. The authors discuss the possible relationship with rat-bite fever.

The occurrence of sporadic or epidemic cases of infectious erythema should be watched for; perhaps after fuller investigation of the varieties of this vague group, the original name may some day be left without an owner.

THE NATURE OF MYXŒDEMA.

THYREOID deficiency was first described when clinical observation occupied a more prominent place in the field of investigation of disease. It is natural, therefore, that in those days the features which caused Ord in 1878 to coin the name "myxœdema" were curious subcutaneous swellings observed in some adults suffering from thyreoid deficiency. More recently attention has been turned from these not very common external manifestations, and the mind of the investigator of today turns rather to the lowered basal metabolic rate and the increase in the blood cholesterol, somewhat, it must be admitted, to the detriment of a clear clinical picture. But it is known that thyreoid substance has some effect in causing dehydration, and this causes us to ask some questions: Is there stored fluid either within or without the body cells in myxœdema? When dehydration occurs after administration of thyroxin, does the water come from the cells themselves or from the intercellular spaces? And has the well known effect of thyroxine in increasing protein usage in the body any bearing on this water problem? Knowledge on these points would shed some light on the nature of myxœdema itself, and a contribution to such knowledge is made by F. B. Byrom in a study of the effect of intravenous administration of thyroxin.[1]

Byrom found that in a normal subject there was a loss both of protein and body water following the injection of thyroxin. The water contained more potassium than sodium and, therefore, presumably came from the intracellular stores, which, of course, were also the source of the protein. The subjects of experiment were kept at rest in a metabolism ward, diets were weighed and analysed for total nitrogen, potassium and sodium, and the stools and urine were also analysed. Care was taken to control the amount of sodium chloride ingested. The myxœdematous subjects were likewise found to suffer loss of both protein and water after thyroxin was given; but in this case there was excess of sodium over potassium, pointing to an extracellular source for much of the water. The water loss appears to be bound up with the protein loss, thus indicating an abnormal accumulation of some protein in the extracellular compartments of the body in hypothyreoid patients. It is on this extracellular protein that the effect of thyroxin in myxœdema seems chiefly, or at least primarily, to operate. Byrom considers that this protein has sufficient colloidal osmotic pressure to divert saline solution from the plasma to the interstitial fluids. He points out that in this way we can account for the curious nature of the myxœdematous swellings, and also for the fact that the plasma is low of volume but relatively high in protein content. One very interesting point about this work is that it brings us back to Ord's original clinical concept of myxœdema, by which it was assumed that there was an excess of mucoprotein in the tissues. It must be admitted that many clinicians have recently been inclined to rebel against the more popular assumption that myxœdema was merely an expression of lowered basal metabolism. That there are qualitative as well as quantitative changes seems evident, and the parallel with hyperthyreoidism will be recalled, for in this disorder of metabolism also there are forces at work more complex than a simple raising of the metabolic rate.

It is of interest also to refer here to the use of thyreoid substance in nephrosis. The extraordinary tolerance of nephrotic patients to thyreoid has been noted by many, and as the blood cholesterol is raised both in nephrosis and myxœdema, it has been suggested that this alteration in blood chemistry is responsible for the readiness with which thyreoid is tolerated. R. S. Aitken[1] has just proved that this is not so, for he has administered thyroxin to a patient suffering from cirrhosis of the liver, in whom there was a considerable hypercholesterinæmia, and

[1] *Clinical Science Incorporating Heart*, Volume I, Number 3, 1934.

failed to demonstrate any unusual degree of tolerance. Thus excess of cholesterin does not inhibit the action of thyroxin.

In conclusion, to return to a consideration of the fundamental nature of myxœdema, it is interesting to speculate on a suggestion of Byrom. He regards myxœdema as an attempt on the part of the body tissues to establish their embryonic environments by flooding the intercellular spaces with muco-protein. This speculation is not of itself of much practical importance, but it helps to divert our attention from a purely mathematical expression of an endocrine disorder that surely must be both complex and far-reaching.

THE ONSET OF BACTERIAL ENDOCARDITIS.

There are many things known about that very serious and usually fatal disease, bacterial endocarditis, but some are as yet unknown. The exact portal of entry of the infecting organism is a matter of great importance, for it is possible that if this were more accurately understood prophylaxis might be within reach. In order that this type of cardiac infection may occur, it is almost an essential that there be a preexisting chronic lesion, for example, of congenital or rheumatic origin. Therefore it is pardonable to digress briefly to consider if it is always possible to diagnose this preexisting lesion. It is curious that such a condition as auricular fibrillation well nigh precludes the possibility of a supervening streptococcal infection of the heart, but apart from this is there any difficulty in recognizing existing valvular disease? The congenital forms are usually easily picked up, even if not readily distinguished, but it would appear that it is not always simple to exclude the presence of rheumatic cardiac infection.

Frank Hawkins has collected a series of cases in which there was no recognizable sign of clinical heart affection and yet indubitable evidence of rheumatic carditis was found at autopsy.[1] In some of these the evidence was found in the heart muscle only, in the form of Aschoff bodies; in the remainder the endocardium was the site of the usual and familiar changes in the customary parts. Death occurred in all these as the result of other disease in no way related to the cardiac lesions. It is of great interest to ponder on the quoted figures: of the 96 cases in which rheumatic heart disease was found *post mortem*, 17 were entirely unsuspected from the history and clinical findings. That ten patients should show undoubted Aschoff bodies in the myocardium and that seven should have evidence of mitral disease without these facts being known during life is instructive. This being so, it will be seen that perhaps the percentage of patients mentioned in text books as contracting bacterial endocarditis without previous rheumatic heart disease may have to be reduced.

Turning to the question of the actual genesis of the more severe type of endocardial disease—that due to infection by one of the pyogenic bacteria—it is of interest to study the results of a research carried out by Harry Weiss on the records of 364 patients who were admitted to the Mount Sinai Hospital, New York, suffering from this complaint.[1] Weiss states that the outstanding feature of the histories is the association of the onset with an attack of some infection of the upper respiratory tract. Influenza and tonsillitis were of particular importance, as has been noted by other writers. The similarity in strains of the streptococci found in the heart vegetations or in the blood stream to those found in the upper air passages has also been observed. Weiss, in discussing the literature, comes to the conclusion that with some few, though probably genuine exceptions, it is the soil of the heart tissues previously damaged by congenital, syphilitic or rheumatic disease that is preferred for the implantation of a streptococcal infection. As regards the actual preceding event which seemed to determine the settling of the streptococci on the endocardium, Weiss presents several interesting case histories in which this event appears to be a "cold", influenza, tonsillitis, tonsillectomy, or the extraction of a septic tooth or teeth. It would appear likely that these varieties of trauma in the upper air passages can set up a transient bacteriæmia, which is no doubt much more common than we think, and in the presence of an existing lesion it is easy to see how implantation may occur. In one of his cases the patient had previously had splenectomy performed for thrombocytopenic purpura; probably this had some bearing upon the patient's subsequent lowering of resistance. Weiss also gives instances in which it would seem possible that the portal of entry was the genito-urinary tract, the middle ear or a wound. These considerations lead to the conclusion that although the rôle of the septic focus has no doubt been exaggerated in certain of the more chronic diseases, there is no question that we should be cautious in exposing the possessors of known or suspected cardiac lesions to the risks of bacteriæmia. Perhaps the general health and resistance of a patient, as judged by the rough clinical standards of well-being, blood state, blood pressure, appetite *et cetera*, are too often overlooked, for it is obvious that if any person has to be subjected to any surgical ordeal, the best time is not one of depressed health. Also, in the case of a child with a congenital heart lesion, special care should be given to the mouth and throat in order that the chance of some extraneous infection by streptococci should be minimized. It is true that it would be difficult by any statistical survey to prove the efficacy of any such attempts to practise preventive medicine, but the general practitioner may comfort himself that he is likely to do much more good in this simple way with his patients who are the subjects of rheumatic or other forms of cardiac disease than the specialist who establishes an obscure diagnosis of bacterial endocarditis.

[1] *Archives of Internal Medicine*, November, 1934.

Abstracts from Current Medical Literature.

SURGERY.

Cholecystitis Without Stone.

W. A. MACKEY (*The British Journal of Surgery*, October, 1934) records the results of the investigation of a large series of cases of cholecystitis without stone; and after discussing the clinical, radiological and pathological aspects thereof, makes an attempt to determine the factors of value in estimating prognosis. An analysis of nearly all the available statistics for the last ten years for simple cholecystectomy, representing over six thousand cases of wide geographic distribution, reveals that the general experience of the surgeons concerned would lead them to expect cure or improvement in nearly 90% of cases of cholelithiasis and in rather more than 80% of cases of cholecystitis without stone. After a preliminary survey of the series it is suggested that in cases of cholecystitis without stone one may expect an operative mortality of about 3%, cure of symptoms in 30%, improvement in 30%, and an unsatisfactory result in 37%. The remainder of the paper consists of efforts to determine the characteristic features of cases suitable for cholecystectomy, and the author concludes that cholecystitis without stone is a condition on the borderline between functional and organic disease and that no single test is infallible. To establish a diagnosis of cholecystitis, the history should be characteristic and include biliary colic, in which case a very high percentage of satisfactory results may be expected. The analyses give no support to the theory that either flatulent dyspepsia or food selection necessarily indicates a diseased gall-bladder and that these symptoms will be relieved by operation. With regard to radiography, caution is required in the interpretation of reports, as impairment of the density of the shadow is not always associated with a pathological gall-bladder or a happy end result. The series indicated that the results of surgery in cases of cholecystitis without stone are relatively unpredictable in the individual case, in spite of the most modern laboratory aids.

Peptic Ulcer.

G. B. EUSTERMAN AND J. G. MAYO (*The American Journal of Surgery*, October, 1934) draw attention to the occurrence in exceptional circumstances of chronic ulcer of the stomach following either external, non-penetrating injuries of the epigastrium, or trauma from within, as in congenital diaphragmatic hernia or from foreign bodies within the stomach. Such ulcers frequently lack much induration and are usually amenable to treatment. The authors quote four postulates which claimants for compensation should satisfy, namely: proof of the absence of gastric disease prior to the accident, proof that the trauma was confined to the epigastrium and was sufficient to cause the lesion, proof that the symptoms followed the injury immediately, and demonstration of a continuation of the symptoms and signs pointing to gastric or duodenal ulcer.

Urological Complications in Spinal Injuries.

J. F. CONNORS AND I. E. NASH (*The American Journal of Surgery*, October, 1934) review the literature relating to injuries of the spine, with special reference to the mortality and morbidity figures in both military and civil practice. After providing a brief description of the pathological physiology of the spinal cord and bladder after such injuries, they find that infection of the urinary tract is responsible for the greatest number of deaths, and that this infection is introduced during catheterization. Presenting a series of 54 cases without a single instance of infection of the urinary tract, the authors stress the necessity for the avoidance of catheterization. In minor injuries the patient may void urine voluntarily after encouragement, and in the more severe lesions a policy of "watch and wait" is employed. Before distension becomes pronounced, manual suprapubic pressure is applied, and it is claimed that this is very efficient, but may not be tolerated if the distension has been neglected too long. In some cases the bladder is allowed to empty by overflow, care being taken to prevent bedsores. It is stated that rupture of the bladder will not occur in these circumstances. When the above schemes fail, suprapubic cystostomy is employed.

Volkmann's Syndrome.

P. MATHIEU *et alii* (*La Presse Médicale*, Number 92, November, 1934) report a case of Volkmann's syndrome and suggest arteriectomy or periarterial sympathectomy for this condition. After mentioning that Leriche and others have drawn attention to the frequency with which brachial arterial obliteration is encountered in Volkmann's paralysis, the authors report a case history, together with the results of the histopathological examination of the affected vessels. They point out that the symptoms are due to two factors, namely, the damage to the artery, which may result in obliteration thereof, and irritation of the sympathetic nerves accompanying the brachial artery. The results of sympathetic irritation are most pronounced in the hypertonus of the flexor group of muscles. The authors conclude by advocating that in early cases the brachial artery should be exposed just above the elbow and submitted to close examination; if, after reposition of bony fragments *et cetera*, the artery fails to pulsate or is thrombosed, then the affected portion should be excised, the anastomotic circle being left about the elbow to carry on the circulation. In those early cases when the artery is found to pulsate quite well, a periarterial sympathectomy is advised to minimize the hypertonus of the flexors. Even in advanced or delayed cases operation is advocated, and here it is claimed that periarterial sympathectomy provides grounds for a better prognosis.

Empyema.

RALPH F. HARLOE (*The American Journal of Surgery*, November, 1934) states that the frequency of empyema is greater than is generally supposed or recognized. It sometimes follows penetrating injuries, rupture of a lung abscess, or surgical puncture. Only rarely does it spread from subphrenic or mediastinal suppurations. The so-called "unresolved" pneumonia generally means suppuration. Empyema may be caused by infection of a pleural effusion due to the rupture of a small subpleural abscess. It may be due to the direct extension of the infectious process from the lung, or possibly the infection is blood-borne. Experimentally it has been shown that the presence of blood in the pleural space is an important factor in the production of empyema. Careful technique is most important when introducing an exploring needle. Empyema is reported in an infant of ten days. In all protracted pneumonias the presence of empyema should be suspected. In influenza epidemics effusion sometimes occurs within seventy-two hours, and the effusion may be almost immediately infected. Two chief methods of treatment are by repeated intercostal puncture or by costectomy. Several authors have stressed the fact that a patient with empyema is never cured until the resulting cavity has been obliterated. Packard has reported a series of 50 children treated with closed drainage and believes that this method meets its greatest indication in children. Danna has recently reported 35 cases treated by periodic aspiration with air replacement. The author believes that in very young infants it is imperative that the pleural cavity be freed entirely of pus and kept free by some irrigation method. The general condition of the patient must be the first guide in deciding when to operate. The writer has employed a closed negative pressure operation with irrigation in all types of empyema, and it has not been necessary to perform costectomy for the treatment of acute empyema in any of these cases. An exploratory thoracentesis is performed and the depth of the pus from the surface is noted. A small stab wound is made through the skin and a trocar and cannula suited to the size of the patient are passed through the chest wall. A special catheter is passed through the cannula and the cannula is withdrawn. Pus is aspirated slowly by means of a syringe. Not more than 150 cubic centimetres are

removed at one sitting and tends to prevent shock. Irrigation is then commenced with normal saline solution. At the end of twenty-four hours Dakin's solution is substituted. Irrigations are repeated every two hours during the day and every four hours during the night. The clamp is never removed from the catheter, excepting during irrigations. The tube is removed in from two to six weeks, depending on the patient's temperature and the quality of the washings from the pleural cavity. The author claims that this method is devoid of shock. It may be employed in any type of empyema in which the pus is accessible to the aspirating needle. Costectomy for the relief of chronic empyema will never be necessary if post-operative supervision is adequate. The method is said to be safe in bilateral empyema. Collapse of the lung does not occur. If this method is employed in infants it is not necessary to wait for the clear effusion to change into thick pus.

Death from Appendicitis.

CHARLES GORDON HEYD (*The American Journal of Surgery*, November, 1934), writing on deaths from appendicitis, states that the presence of pus, gangrene or general peritonitis is evidence of neglect or delay of treatment. The author believes that the administration of aperients is at the head of the list of the causes of death in this disease. In 88% of persons the blood supply to the appendix is essentially a terminal circulation. The author claims that infection commences in the mucosa in almost 99% of cases. The small empyema which results may terminate in one of three ways, namely, drainage back into the intestine, by perforation, or else by a combined suppurative and gangrenous process. The sudden onset of acute cramp-like abdominal pain is the first and most important warning symptom of appendicitis. Of 402 patients who had acute appendicitis and who did not take a laxative, one out of 80 died; among 992 who took one laxative, the mortality was one in 14; and among 992 patients who took two or more laxatives, one out of seven died. There is only one treatment for appendicitis, and that is surgical intervention. No food or drink should be taken after the onset of symptoms. Any laxative is strongly contraindicated, and the patient must be kept at rest until diagnosis and treatment are determined. The surface area of the peritoneum is said to be almost equal to the surface area of the body of the individual, and under certain circumstances may absorb 8% of the body weight in one hour.

Perinephritic Abscess.

ARTHUR M. DICKINSON (*The American Journal of Surgery*, November, 1934) discusses perinephritic abscess. This condition was recognized by Hippocrates, who advised early drainage. Infection of the perinephritic space may occur from direct extension of the kidney, or it may be embolic. Frequently the condition is not suspected until the patient is moribund. Reports from the Mayo Clinic show that the average time lapsing from the onset of symptoms to the establishment of correct treatment has been thirty-eight days. A primary focus of infection is not always obvious. Frequently the condition arises from an inflamed appendix having some portion retroperitoneally placed. Ulcerations of the intestines are also a common cause. Pelvic infection has been responsible in the female. The amount of pus present may be great or small, and the condition is occasionally bilateral. The symptoms are often indefinite and the condition may be confused with influenza or typhoid fever. Peritonitis is sometimes simulated. Fever and profuse sweating are common at the onset. Pain is variable in site and degree. Urinary tract symptoms are not common. Radiography is frequently helpful. Incision and drainage are usually followed by dramatic results. Any pockets present must be obliterated at the time of operation. No attempt should be made to deal with the primary cause at the time of draining the abscess. Three cases are reported in detail in which the appendix appeared to have been the primary focus of infection.

Status Lymphaticus.

DOUGLAS SYMMERS (*The American Journal of Surgery*, October, 1934) records that *status lymphaticus* was encountered 240 times in a series of 4,000 autopsies at the Bellevue Hospital, New York, representing an incidence of 6·2%. In this series the condition occurred in males six times oftener than in females. This author presents the material not so much to emphasize the occurrence of sudden death in *status lymphaticus*, but rather to impress the signs by which the condition may be detected or suspected in the living patient, together with certain associated disturbances which are sometimes overlooked. The condition is defined as "a combination of hereditary constitutional anomalies, entering into which, among other things, are certain peculiarities of configuration, with preservation or even hyperplasia of the thymus at an age when involution is to be expected, hyperplasia of the lymphoid cells in the spleen, intestines and elsewhere, changes in the distribution of the hair and hypoplasia of the vascular system". Records show that while individuals with *status lymphaticus* may enjoy a long life, sudden death often occurs as a result of trivial factors, possibly anaphylactic. The external features which are claimed to be suggestive of *status lymphaticus* are the delicate texture of the dead white, pasty or creamy skin, the gracefully proportioned body, the hypoplasia of the genitals, and the tendency to changes in the distribution of hair, especially over the pubes. The author remarks upon the absence of obvious lymphoid hyperplasia in the inguinal, axillary, mesenteric and cervical glands, and states that in 50% of cases the faucial, lingual and pharyngeal tonsils are enlarged, while hyperplasia is found in the intestinal follicles in 80%. Among the associated conditions mentioned, the occurrence of lymphocytosis in Graves's disease is emphasized and its prognostic importance recalled.

The Thyreoid Gland of Hyperthyreoidism after Surgical Treatment.

WM. FRANCIS RIENHOFF, JUNIOR (*The Western Journal of Surgery, Obstetrics and Gynecology*, October, 1934) discusses the histological structure of the thyreoid gland in patients regarded as cured of hyperthyreoidism by surgical treatment. Horsley and Halstead, in the last quarter of the nineteenth century, demonstrated alterations in the structure of the thyreoid gland as being due to a hypertrophy and hyperplasia of its parenchyma. In 1923 Rienhoff demonstrated certain microscopic changes of the structure of the gland following the administration of iodine. The author has made an attempt to study the histological pattern of the remaining stump of the thyreoid following operation for exophthalmic goitre. Patients have remained entirely well following operation. Four cases are reported. All four patients recovered completely from the signs and symptoms of hyperthyreoidism following operation. The portions of gland left at operation retained their microscopic structure, but did not undergo any increase in the gross size of the tissue. Two patients had notably unstable nervous systems, and in them the hypertrophy and hyperplasia were more pronounced. The view is advanced that the original hypertrophy and hyperplasia represent some form of benign overgrowth which should be classified as a benign tumour. Hyperthyreoidism is known to occur in certain cases of carcinoma of the thyreoid.

The Use of Bacteriophage in Colitis.

W. J. MACNEAL, F. C. FRISBEE AND M. APPLEBAUM (*Archives of Surgery*, November, 1934) report the use of bacteriophages in chronic colitis of undetermined causation and in intestinal fistulæ. Of fourteen patients with colitis, seven manifested definite clinical improvement, in six cases the result of treatment was not discoverable, and one patient obtained no relief. In two instances the authors obtained satisfactory results by using bacteriophage in the treatment of post-operative intestinal fistulæ. They describe the treatment as empirical and point out that the results are not dramatic. Since the treatment is harmless the authors think that it should sometimes be tried.

Special Articles on Treatment.

(Contributed by request.)

XLVI.
THE TREATMENT OF TETANUS.

Prophylactic Treatment.

PROPHYLACTIC treatment consists, first, in the early treatment of all contaminated wounds and, secondly, in the administration of tetanus antitoxin.

As soon as possible after infliction of the wound it should be excised and all foreign bodies, soil, damaged and devitalized tissue should be removed, more particularly muscular tissue which does not contract on contact with the dissecting forceps. The wound should be left open and not tightly sutured.

As regards the so-called "splinter" wound, and especially the punctured wound of the sole of the foot, caused by standing on a nail or a large splinter of wood, excision of this type of wound is indicated either under general or local anæsthesia, and the wound should not be sutured.

A prophylactic subcutaneous injection of tetanus antitoxin should be given immediately, preferably 3,000 international units of a concentrated antitoxin. The use of concentrated antitoxin diminishes the risk of anaphylaxis. Serum is now available containing from 1,000 to 3,000 or more units per cubic centimetre. When the wound has been grossly contaminated with soil, the dose should be repeated at intervals of a week until four doses have been given. The reason for the repetition of the dose is that passive immunity is of relatively short duration.

Treatment of Declared or Acute Tetanus.

The treatment of declared or acute tetanus may be considered under three headings: (*a*) specific treatment by tetanus antitoxin, (*b*) sedatives and general care of the patient, (*c*) the treatment of the wound.

Antitoxin Treatment.

It cannot be too strongly urged that serum treatment should be immediately instituted in all cases of acute tetanus or even of suspected tetanus. It is of interest to note that during the War the following directions were issued by the "Adviser in Pathology", Headquarters, Lines of Communication:

1. Give large doses of antitetanic serum.
2. Give them early and persistently.
3. Use the intrathecal route up to its limit and supplement by additional doses given intravenously and hypodermically.

Since that time the manufacturers of tetanus antitoxin have produced much more highly concentrated serum. This reduction in the volume of the serum has made it possible to give a much greater number of units, particularly by the intrathecal route. So far as the method of administration is concerned, there is not much to be added to the directions quoted above. As regards the intrathecal route, the majority of writers advocate this in conjunction with the intravenous and subcutaneous and intramuscular routes, and draw attention to the results obtained by Sherrington in the treatment of tetanus experimentally produced in monkeys.

In recent literature a note of caution is sounded by some writers concerning antitoxin given intrathecally. Some regard it as dangerous and others claim that they obtain as good results by means of antitoxin administered intravenously or intramuscularly.

Method and Dosage.—It is advisable to use concentrated serum. Lately in some quarters there has been a tendency to administer enormous doses of antitoxin.

Intrathecal Route by Lumbar Puncture.—It is wise to administer a general anæsthetic, such as ether, when doing a lumbar puncture. Ten to twenty cubic centimetres of cerebro-spinal fluid are withdrawn and an equal volume of the most potent antitoxin available, which has been warmed, is injected. The foot of the bed should be raised for at least half an hour after the injection.

By means of concentrated serum 30,000 to 60,000 or more international units can be given in the manner indicated.[1]

Intravenous, Intramuscular and Subcutaneous Routes.—The above dosage should be supplemented by at least 30,000 international units given intravenously and 30,000 international units given intramuscularly or subcutaneously. The dosage quoted above should be regarded as the minimum to be administered during the first twenty-four hours after treatment is begun. Should lumbar puncture be impossible, an additional dose, 60,000 units, should be given intravenously or intramuscularly.

Very much larger doses have been given by some writers, who use more particularly the intravenous and intramuscular methods. Fifty thousand to one hundred thousand international units should be given daily by the intravenous, intramuscular or subcutaneous route and continued until the symptoms abate, when the amount of the daily dosage should be reduced. The serum should be warmed and, when given intravenously, may be mixed with an equal volume of normal saline solution.

Sedatives and General Care.

Death is caused in tetanus by gradual cardiac failure, œdema of the lungs, or asphyxia during a spasm. It is therefore highly desirable that in order to control the exhaustion suitable nourishment should be given. In severe cases this will consist of fluids. It is imperative that the patient be placed in a quiet and darkened room and that some sedative should be administered, such as sodium bromide, 2·0 grammes (thirty grains), every four hours until the spasms are controlled. Morphine is usually required.

Other sedatives which may be used in appropriate doses are chloral hydrate and "Sodium amytal". Possibly paraldehyde given by the rectum in doses of 4·0 cubic centimetres (sixty minims) for every stone of body weight, in 180 cubic centimetres (six fluid ounces) of normal saline solution, would prove to be a useful sedative. Ether or chloroform may be required to control spasms.

Treatment of the Wound.

The wound of infection may be very small or even an apparently healed wound. Some writers advise the excision of the apparently healed wound. In cases of established tetanus it is wise not to interfere with the wound until the patient is well saturated with tetanus antitoxin, as there is a distinct danger of liberating a fatal dose of toxin. One remembers the cases of tetanus following secondary operations on gunshot wounds many weeks after the original wound had been inflicted. It was to counteract this danger that instructions were given for the administration of a dose of antitoxin before subsequent operation. Injection of serum around the wound has been advocated; should this be done, it should be given well beyond the granulation tissue surrounding such a wound.

In the case of a wound of a limb it may be possible to inject the trunk of the main nerve to the part with antitoxin. When the patient is well saturated with antitoxin it is advisable to see that the wound is adequately drained. The wound may be dressed with hypertonic saline solution, Dakin's solution or peroxide of hydrogen. Carrel's tubes, by means of which the wound can be alternately flushed with peroxide of hydrogen and Dakin's solution, have proved very satisfactory.

Amputation should be performed only if justified by other indications.

Anaphylactic Shock.

A number of patients are hypersensitive to horse serum, and anaphylactic shock may follow injection of serum, particularly intravenous injection. Should a patient prove hypersensitive, he should first be desensitized. For the prevention of anaphylactic shock atropine sulphate is recommended—0·65 milligramme (one-hundredth of a grain) given subcutaneously half an hour before the serum is administered. Should anaphylaxis occur, adrenaline chloride, half to one cubic centimetre of 1 in 1,000 dilution, should be given intramuscularly.

GEORGE BELL,
Honorary Surgeon, Sydney Hospital.

[1] The international unit is half the United States of America unit.

British Medical Association News.

MEDICO-POLITICAL.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on February 6, 1935, DR. RUPERT M. DOWNES, the President, in the chair.

Financial Statements.

DR. C. H. MOLLISON, the Honorary Treasurer, presented the statement of receipts and payments of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria for the period January 11, 1934, to January 10, 1935. The statements, which are published herewith, were adopted.

BRITISH MEDICAL ASSOCIATION—VICTORIAN BRANCH.

Statement of Receipts and Payments for Period from January 11, 1934, to January 10, 1935.

RECEIPTS.	£ s. d.	£ s. d.
January 11, 1934—		
To Balance, National Bank of Australasia, Limited	354 16 6	
„ Cash in Hand	12 0 0	
		366 16 6
January 10, 1935—		
„ Subscriptions—		
Metropolitan Members	3,502 4 9	
Country Members	1,554 10 0	
Junior Members	242 12 6	
		5,299 7 3
„ Duplicating Department		50 8 7
„ Hire of Lantern		1 10 0
„ Sale of Catalogues *et cetera*		1 17 6
„ Balance, National Bank of Australasia, Limited		2 7 4
		£5,722 7 2

PAYMENTS.	£ s. d.
January 10, 1935—	
By Medical Society of Victoria	2,438 10 4
„ *The British Medical Journal*	1,587 2 3
„ Rebates to Subdivisions	12 13 6
„ Medical Benevolent Fund	3 0 6
„ Salaries *et cetera*	1,141 12 3
„ Postages, Duty Stamps *et cetera*	129 3 6
„ Printing	40 9 10
„ Stationery	69 11 1
„ Commission	9 2 2
„ Advertising	15 13 0
„ Audit Fee	5 5 0
„ Telephone	9 13 4
„ Repairs *et cetera*	3 17 0
„ Bank Charges *et cetera*	1 10 10
„ Electric Light and Power	6 5 1
„ Federal Council Expenses	127 2 0
„ Lanternist	5 10 0
„ Sundries	23 12 6
„ Travelling Expenses	62 13 0
„ Purchase of Duplicator	18 0 0
„ Cash in Hand	12 0 0
	£5,722 7 2

Compared with the Books and Accounts of the British Medical Association (Victorian Branch) and found to be in accordance therewith.
February 2, 1935.
J. P. MAJOR, Honorary Secretary.
C. H. MOLLISON, Honorary Treasurer.
J. V. M. WOOD AND COMPANY, Chartered Accountants (Aust.)

MEDICAL SOCIETY OF VICTORIA.

Statement of Receipts and Payments for Period from January 11, 1934, to January 10, 1935.

RECEIPTS.	£ s. d.
January 10, 1935—	
To Transfer from British Medical Association, Victorian Branch	2,438 10 4
„ British Medical Insurance Company (Loan)	500 0 0
„ Sundry Contributions—C. Stanton Crouch Presentation	8 11 0
„ Donation	3 3 0
	£2,950 4 4

PAYMENTS.	£ s. d.
January 11, 1934—	
By Balance, National Bank of Australasia, Limited	104 4 5
January 10, 1935—	
„ THE MEDICAL JOURNAL OF AUSTRALIA	1,254 7 6
„ Interest on Building Fund Debentures	421 15 0
„ Hospital Benefits Association (Loan)	500 0 0
„ Salaries—	
Library Clerk £72 0 0	
Caretaker 100 0 0	
	172 0 0
„ Library, Journals and Binding	182 11 11
„ Rates, Taxes and Insurance	28 13 10
„ Light and Power	7 5 2
„ Audit Fees	5 5 0
„ Telephone	31 0 9
„ Bank Charges	1 11 1
„ Repairs	18 15 0
„ Sundries	18 4 2
„ Furniture	22 1 0
„ Balance, National Bank of Australasia, Limited	182 9 6
	£2,950 4 4

Compared with the Books and Accounts of the Medical Society of Victoria and found to be in accordance therewith.
February 2, 1935.
J. P. MAJOR, Honorary Secretary.
C. H. MOLLISON, Honorary Treasurer.
J. V. M. WOOD AND COMPANY, Chartered Accountants (Aust.)

Medical Societies.

THE MEDICAL DEFENCE SOCIETY OF QUEENSLAND.

THE annual meeting of the Medical Defence Society of Queensland was held at the B.M.A. Building, 35, Adelaide Street, Brisbane, on February 14, 1935, DR. ALEX. MARKS, the President, in the chair.

Annual Report of Council and Financial Statement.

The annual report of the Council and the financial statement for the year ended December 31, 1934, were presented and adopted on the motion of the President, seconded by Dr. A. B. Carvosso.

The report and financial statement are as follows:

The Council has pleasure in presenting the following report of the Society for the year ended December 31, 1934.

Membership.

The total membership of the Society is 356, exclusive of 22 members whose subscription for the current year had not been paid by December 31.

During the year 17 new members were elected and two members rejoined. Our losses have been as follows: Eleven members have left the State, three resigned and four deceased.

We regret to record the following losses by death: Dr. P. S. Clarke, Dr. Frederick Challands, Dr. Cecil Uren, and Dr. R. A. Meek.

Office-Bearers and Council Elected for 1934.

President: Dr. A. H. Marks.

Vice-President: Dr. Donald A. Cameron.

Honorary Secretary: Dr. Neville G. Sutton.

Honorary Treasurer: Dr. R. G. Quinn.

Councillors: Dr. A. G. Anderson, Dr. A. B. Carvosso, Dr. G. W. Macartney, Dr. S. F. McDonald, Dr. W. N. Robertson, Dr. Kerr Scott, Dr. H. S. Waters.

Medico-Legal.

Four cases were submitted to the Council by members during the year, three of which were referred to our solicitors for legal advice. Two cases have been finalized and one case is still pending. The fourth case did not come within the scope of the activities of the Society.

Amendment of Articles of Association.

A special general meeting of the Society was held on August 21, 1934, when the amendment of the Articles of Association was approved. Books of the Memorandum and amended Articles of Association have been issued to members of the Society.

Registration of Membership.

The limit of the number of members of the Society for registration purposes has been increased to 600.

Finance.

The total assets of the Society are £4,097 2s. 5d., of which £2,882 3s. 6d. is invested in Australian Consolidated Treasury Bonds and £509 7s. 6d. in Australian Consolidated Inscribed Stock.

The surplus income over expenditure for the year amounted to £270 6s. 6d. The total expenditure amounted to £95 17s. 10d., of which the sum of £40 2s. 6d. was for legal costs. The amount received for entrance fees and subscriptions was £214 15s. 9d., and the amount of income from investments totalled £149 5s. 10d.

ALEX. MARKS,
President.

Election of Councillors.

Dr. D. A. Cameron, Dr. S. F. McDonald and Dr. R. G. Quinn, who had retired in accordance with the by-laws of the Society, were unanimously reelected as members of the Council for the ensuing yeaer.

Election of Auditor.

Mr. R. G. Groom, Chartered Accountant (Aust.), was reelected Auditor.

Post-Graduate Work.

GENERAL REVISION COURSE, 1935.

THE New South Wales Permanent Post-Graduate Committee announces that the general revision course for 1935 will commence on Monday, May 27, and end on Friday, June 7, 1935. Railway concession tickets are available to those attending the course, for their wives and children under sixteen years of age, also for daughters who are wholly dependent on them.

Instruction will be given at hospitals as follows:

Monday, May 27.—Sydney Hospital.
Tuesday, May 28.—Royal Alexandra Hospital for Children.
Wednesday, May 29.—Royal North Shore Hospital of Sydney.
Thursday, May 30.—The Prince Henry Hospital.
Friday, May 31.—Crown Street Women's Hospital.
Saturday, June 1.—Sydney Hospital.
Tuesday, June 4.—Royal Prince Alfred Hospital.
Wednesday, June 5.—Saint Vincent's Hospital.
Thursday, June 6.—Morning: Royal Hospital for Women; afternoon: Tresillian Mothercraft Training School.
Friday, June 7.—Lewisham Hospital.

The fee for this course is £3 3s. Registration and applications for concession tickets should be made to the Honorary Secretary, New South Wales Permanent Post-Graduate Committee, 225, Macquarie Street, Sydney.

For those attending the course the fee for the evening lectures by Dr. Kellaway, previously announced in this journal, will be £1 1s. Cheques should be crossed and exchange should be added to country cheques.

WEEK-END POST-GRADUATE COURSE AT TAMWORTH.

As previously announced, the New South Wales Permanent Post-Graduate Committee will hold a week-end course at Tamworth, New South Wales, in conjunction with the Northern District Medical Association, commencing on Saturday, April 6, 1935. The programme is as follows:

Saturday, April 6, 1935, at the Ambulance Station, Marius Street, Tamworth.

11.30 a.m. to 12.15 p.m.—"General Principles of Diet in Disease", Dr. A. J. Collins.
12.20 to 1 p.m.—"The Treatment of Burns and Scalds", Dr. R. B. Wade.
1 to 2 p.m.—Luncheon at the Royal Hotel, Tamworth.
2 to 2.30 p.m.—"Cardiovascular Degeneration and the Significance of Blood Pressure", Dr. A. J. Collins.
3 to 3.30 p.m.—"Supracondylar Fracture of the Humerus", Dr. R. B. Wade.
4 p.m.—Afternoon tea.
4.30 to 5 p.m.—"Asthma", Dr. A. J. Collins.
5 to 5.30 p.m.—"Acute Osteomyelitis", Dr. R. B. Wade.
6.30 p.m.—Dinner at the Royal Hotel, Tamworth. (All visitors to be guests of the Tamworth medical practitioners.)

Sunday, April 7, 1935, at the Tamworth District Hospital.

9 to 10.45 a.m.—Operations by Dr. R. B. Wade on selected cases.

10.45 to 11 a.m.—Morning tea.

11 a.m. to 12 noon.—Clinical demonstration by Dr. A. J. Collins on selected cases.

12 noon to 12.30 p.m.—Demonstration by Dr. Kathleen Clemens on blood typing and transfusion.

The fee for this course will be £1 1s. Those intending to be present should communicate with Dr. A. Brydon, Honorary Secretary, Northern District Medical Association, Armidale.

Correspondence.

RECOVERY AFTER TAKING OVER 45 GRAINS OF "LUMINAL".

SIR: I thought the following case may be of interest to you.

On January 16 Mrs. M. took between 45 and 50 grains of "Luminal" in one dose. She is a victim to migraine and took the dose regardless of the consequences. She is aged forty-five.

The dose was taken about 3 p.m. on the 16th, but I was not called until 5.30 the following morning, when she was in what her husband described as "a beautiful sleep"—actually a coma—from which he had failed to wake her.

The skin was clammy, temperature 96·4° F., pulse 108, muscles relaxed, reflexes absolutely absent, pupils contracted to pin-point and with no reaction to light. The eyes were watery and tears flowed freely. She was slightly cyanosed and breathing was stertorous.

As it was obviously too late for stomach lavage, she was given one-thirtieth of a grain of strychnine and removed to hospital, where the bowel was washed out and other stimulants given. The strychnine was repeated twice at two-hourly intervals. The cerebro-spinal fluid was turbid and under considerable pressure.

The following day she developed a mild jaundice, which persisted for a week. Retention of urine was troublesome, and catheterization had to be resorted to at intervals. Purgatives proved useless, and up to the tenth day the enema was necessary. A slow but progressive return to consciousness occurred, the patient appearing quite normal in speech and thought by the fifth day. Drowsiness, however, persisted till the tenth day.

It would seem that "Luminal" in such massive doses has a certain paralysing effect on the unstriped muscle of the bladder and bowel. In a patient taking "Luminal" regularly for epilepsy I have at times noted some slight difficulty of micturition. This subject may be usefully pursued.

Yours, etc.,

R. M. DUNSTONE, M.B., Ch.B. (Glasgow).

Bogan Gate,
New South Wales,
February 4, 1935.

FRUIT AS A CAUSE OF BOILS.

SIR: Before the days of rapid transport the more perishable tropical fruits were little known in the southern States of Australia. Their effects upon the individual were still less known.

Mangoes were first made known to the writer when a boy by the tales of an old servant who had been in the British Army in India. He described the wonders of this fruit and how too free consumption of it resulted in admission to the military hospital for the treatment of crops of boils. Also that upon discharge therefrom the victim was often so sorely tempted again that readmission to hospital was necessary.

Faith in the old soldier's story was somewhat discredited when many years later there appeared in Manson's "Tropical Diseases", under the heading of "Boils in the Tropics", the following sentence: "Mangoes, probably erroneously, are frequently held responsible."

Again after the lapse of considerable time, the writer, in the course of practice, saw a patient who was suffering severely from boils. It was in the fruit season at Christmas time. Under treatment he duly recovered. The following year again at Christmas time he came with a similar crop of boils. The patient was a well known city business man living a normal existence and had been in perfect health between the two attacks.

Turning away to get an instrument the remark was casually dropped: "If we were in India one would say you had been eating too many mangoes." "Good heavens!" was the reply, "last Christmas I had a case of mangoes sent to me and another one this Christmas." Queensland colleagues may be interested.

Yours, etc.,

F. S. TANGE, M.B., Ch.M. (Sydney).

830, Military Road,
Mosman,
New South Wales,
February 6, 1935.

ALLERGY IN OTO-RHINO-LARYNGOLOGICAL PRACTICE.

SIR: I have read with interest the letters of Dr. Bryan Foster and Dr. Chas. Sutherland concerning "Allergy in Oto-Rhino-Laryngological Practice" in your issue of THE MEDICAL JOURNAL OF AUSTRALIA, February 2, 1935.

I heartily endorse the views they have expressed and as a matter of historic interest would like to point out that as far back as 1920 the late Dr. Frank Andrew realized the futility of operative measures in the treatment of nasal allergy. At that time and at his suggestion I made various experiments and investigations concerning the causative factors of allergy in Australia. The information so derived was utilized by the Commonwealth Serum Laboratories when they first placed on the market allergens for diagnosis and treatment of such conditions in this country.

Yours, etc.,

IVAN MAXWELL.

61, Collins Street,
Melbourne, C.1,
February 9, 1935.

PEDICULOSIS PUBIS.

SIR: Your correspondents, Dr. Minogue of your issue of January 5 and Dr. Ewan of February 9, might be interested in an article I contributed to THE MEDICAL JOURNAL OF AUSTRALIA of February 21, 1925—ten years ago—entitled "Heliotropine: The Best Pediculicide". It seems that heliotropine again needs a little advertisement, since your correspondents do not seem to have heard of it.

In a war-time research carried out for the American National Research Council at the University of Minnesota by Moore and Hirschfelder, heliotropine made a consistent 100% record of successes as a pediculicide for *Pediculus corporis*. A twelve-hour test was the standard. There were some other substances able to make similar records, but heliotropine has special advantages, being non-irritating, pleasantly perfumed and cheap. In my article I mentioned that I had introduced it as an experiment at the Sydney Hospital for *Pediculosis capitis*, that it had rapidly become the only method to be used, and that over a period of five years it had given complete satisfaction, *cito, tute, et jucunde*. The period of use has now covered fifteen years. The ward sisters say it is unnecessary to remove the hair and that one application is enough. The solution is worked liberally into the scalp and washed off after twelve hours.

The formula, heliotropine 5 parts, castor oil 30 parts, and methylated spirit to make 100 parts, is that still in use in the Sydney Hospital. The methylated spirit used is not the ill-smelling variety denatured with pyridine. I notice that approximately the same formula has been incorporated in the A.P.F., the only difference being that the castor oil is made 25% instead of 30%.

I have often wondered whether this heliotropine solution would cure scabies. I never have opportunities for trying it. Perhaps someone with better opportunities would make the experiment.

I have also thought that heliotropine, given as a powder, should prove a good remedy for intestinal worms in animals. It is only slightly soluble in water, but soluble in oils. I have not had any opportunity of testing it as a vermicide.

Yours, etc.,
C. E. Corlette.

175, Macquarie Street,
Sydney,
February 9, 1935.

MAJOR SURGERY AND THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

Sir: The letter of Dr. D. McG. Steele, in your issue of February 2, calls for a reply from me. It calls for a complete reply.

I feel sure that no general practitioners by my letter would accuse me of having made an attack on them because of holding no higher degree than the M.B., B.S., Nor did I wish to imply that any individual with an M.B., B.S. degree only should be debarred from doing major surgical work within his limits. We all know very many such who are very able surgeons, and they have gained such competency because they first became physicians and therefore know their limitations. Why, has not the College of Surgeons recognized their ability by admitting them to membership? My attack is on those juniors who will know no limits and who try to make the public believe that they are surgeons. They are on the increase.

Osler describes a surgeon as "a physician who can operate". From my experience many of the "major operators" could not even be called physicians, because their main concentration is on surgery first.

I made no sweeping condemnation of the general practitioners. Rather did I implore them to help to maintain our profession at its present high level in the public's estimation before it may be too late to do so. Far be it from me to belittle the British Medical Association. I am trying to safeguard it.

Next, Dr. Steele makes an unwarranted attack on the Fellows of the College. He says that they are "*rara aves*" in country districts of this State. I entirely disagree. In the last thirteen years I have covered "almost" every square mile of Victoria in a professional capacity, and I always found a surgeon within reasonable distance everywhere. Some had an M.B., B.S. degree only. They are now F.R.A.C.S.

Next he says: "How many of them would be prepared to undertake a trip of sixty miles in any weather, perform any necessary operation, and be content with a fee of about 25% of that set out in the British Medical Association schedule?"

From my experience of thirteen years I should say all of them. No surgeon whom I have ever called has ever hesitated or refused to come any distance, over any roads, at any time of the day or night. Eleven years ago I practised in Dr. Steele's district. During the winter the roads are the worst in Australia. Great floods were on when I called a Bendigo surgeon to a desperate case which could not be moved. He didn't question fees or roads, he only said he would get there. A Collins Street surgeon who had reached Melbourne at 11 p.m. from another State, started off at once on an eighty-mile car journey to a gravely urgent case, even though I had told him that he could not expect to get more than 25% of his fees—and that was in pre-depression days. Most surgeons with whom I have had the pleasure of being associated, have volunteered their willingness to come to my aid, even though it meant big mileage, in desperate cases, even if it were obvious that there could not be a cent of remuneration for them. I can only conclude that Dr. Steele has not had much experience with surgeons.

Next Dr. Steele writes: "Dr. Peters dons San Benito and makes his obeisance to the College of Surgeons. The reason therefor does not appear very clearly in his letter." I much resent this statement, and I intend to make it very clear indeed. The above remark I also cannot but interpret except as an imputation that an "interested" or over-enthusiastic Fellow (or Fellows) of the College has prompted me to write that letter in its interests. I say most emphatically that not one member of either the medical or surgical profession prompted or instigated me to write that letter. That letter is the result of thirteen years' consideration by me alone. Quite early in my professional career I was anæsthetist to a young M.B., B.S. operator of very little experience. He attempted a major operation which, in my opinion, should be done by a surgeon of at least ten years' experience. The result was disastrous, so much so to my resentful feelings that I was seriously considering "getting out" of the profession and back to commercial life. My mature lay mind and my very immature professional mind clashed very badly. Actually that letter was written by me on the instigation of several exasperated lay people who had real reason therefor. They said to me: "Can this sort of thing not be stopped by the B.M.A.?" "Does it allow it?" and so on. I assured them that I would not let the matter rest. This does not pertain to the above-mentioned case.

The fact that the most eminent men in the profession are only human, and therefore apt to make a mistake, was implanted in me, I think I am safe in saying, before Dr. Steele was born. More or less I have been associated with the medical profession for nearly forty years. It is nearly forty years since I matriculated at the Melbourne University. I then entered the Adelaide University on the arts and science course. I decided on pharmacy as a calling. I had all the necessary subjects except pharmacy to my credit. To get my diploma—that is, before I was permitted by law to dispense a doctor's prescription—I had to go through a four years' apprenticeship. On completion I returned to Melbourne to go on the staff of Messrs. Henry Francis and Company, pharmaceutical chemists, of Collins Street. At the age of thirty-six I entered the Melbourne University for my M.B., B.S. course. This I completed at the age of forty-one, and when I received my degree I was struck by the fact that there was no law to prevent me from doing major surgery—very striking indeed when considering that I had to do a four years' course (to protect the public) for the very simple performance of dispensing a doctor's prescription.

I make no obeisance to the College of Surgeons. I said, in that letter that I had always been in favour of it. Further, I make no recantation, as Dr. B. Cook puts it. What I do regret is that I should have taken part unintentionally in the criticisms against the two clauses in the original constitution of the College. These two clauses were: (i) To educate the public to recognize that the practice of surgery demands adequate and special training. (ii) To safeguard the welfare of the community by indicating that its Fellows have attained a high standard of surgical competency and are of high standing. By no means was I against these two clauses, but I was misled by the supposed "advertising" methods by the College, as were presented to me. I have very good reasons to say that those two clauses should never have been deleted.

Dr. Steele's statement: "Two appendices per thousand *per annum* hardly give sufficient scope for the worst surgeon to ruin his reputation and convince Dr. Peters in the process that a plain degree and surgical competence are incompatible." That statement I will let go for what it is worth; but there should be no such thing as "the

worst surgeon" in our profession. That was precisely the whole meaning of my letter. That is, too, the main object of the College of Surgeons.

Further, Dr. Steele says: "Dr. Peters's suggestion amounts to a proposal to increase the length of the medical course." It certainly does not. But I do suggest that the young graduates, having obtained their M.B., B.S. degrees, should first become physicians, and if they aspire to major surgery, they should have to go through an apprenticeship, so to speak, in the art, to qualify them to practise it. Having first become physicians they will not "rush in to operate where angels shake the head", as Dr. Stewart puts it. And when they have realized how human it is to err, and if they truly have their patient's welfare at heart, they will have their diagnosis confirmed before any surgical intervention.

Dr. Hagenauer writes: "Today the operation . . . without the assistance of consultations to confirm the diagnosis as was the custom a few years ago." And Dr. Hagenauer is one of our oldest and most able physicians and surgeons. (He once came to my assistance in an urgent case in the bush—Glenmaggie Weir, sixty miles. He made no inquiries about his fees *et cetera* first.)

Then, too, as Dr. Stewart puts it: "There would not be an indecent haste . . . lest the patient should be well in the morning."

My letter suggested two things only: (i) That major surgery should be done only by men qualified to do it. (ii) The confirmation of diagnosis before operation. And by no means do I suggest that this must be an absolute 100%. For instance, I was once called upon to do a major operation on the high seas, thirty-six hours from the nearest hospital.

Surely I must have the great majority of the medical and surgical professions behind me on these two points. I certainly would have 100% of public opinion.

Yours, etc.,

A. L. J. PETERS.

Myrtleford,
Victoria,
February 17, 1935.

[This correspondence is now closed.—EDITOR.]

The Royal Australasian College of Surgeons.

GIFT TO THE COLLEGE.

THE following announcement is published at the request of the Executive Committee of the Royal Australasian College of Surgeons.

Dr. Alfred Edward Rowden White, of Melbourne, has donated £1,000 to the building fund of the Royal Australasian College of Surgeons. Dr. White, wishing to add to the beauty of the College building and of the City of Melbourne, expressed the wish that this amount be used to defray the cost of a stone portico as an entrance to the College in Spring Street, Melbourne.

Dr. White feels that the action of the Victorian Government in generously providing such a magnificent site for the College building is without parallel in the medical history of Australia, and that it is an expression of confidence, not only in the Fellows of the College, but also in the whole of the medical profession. Dr. White is desirous that a building standing in such an important position should be dignified and worthy of the medical profession. He wishes his gift to be regarded as much from the point of view of citizenship as from that of membership of the medical profession.

The Royal Australasian College of Surgeons is deeply appreciative of Dr. White's gracious act.

Medical Prizes.

THE HUNTERIAN SOCIETY GOLD MEDAL FOR PRACTITIONERS.

THE rules governing the award of the Hunterian Society Gold Medal for Practitioners have recently been altered.

Any registered general practitioner resident within the British Empire is eligible to compete, and the medal, which is of gold, is awarded annually to the writer of the best essay on a subject selected by the Society. Competitors—men or women—must be engaged in general practice, and essays should be sent in by December 31.

The essay must be unpublished and original, and be based on the candidate's own observation, but it may contain excerpts from the literature on the subject, provided that reference be made to the articles from which they are taken.

A copy of the rules and any further information can be obtained on application to the Honorary Secretary, Mr. Martin Oldershaw, 26, Upper Wimpole Street, London, W.1.

The subject selected for the essay for 1935 is: "The Conduct of Midwifery in General Practice"; and for 1936: "Rheumatoid Arthritis: Its Diagnosis, Treatment and End Results".

NOTICE.

THE Secretary of the Victorian Branch of the British Medical Association announces that the monthly meeting of the Branch will not be held on March 6, as previously announced. The place of this meeting will be taken by a lecture to be given by Dr. Dean Lewis to the Royal Australasian College of Surgeons.

Dr. Lewis's lecture will be delivered on Thursday, March 7, 1935, at the public lecture theatre at the Arts Building, University of Melbourne, at 8.15 o'clock p.m. The title of the lecture will be "Ovarian Hormones and their Relation to Breast Tumours". The lecture is part of the programme of the annual meeting of the Royal Australasian College of Surgeons. All members of the British Medical Association have been invited to attend the lectures and demonstrations in connexion with this meeting.

The inaugural meeting will be held at Wilson Hall, University of Melbourne, on Monday, March 4, at 8.15 o'clock p.m. At this gathering the Syme Oration will be delivered by Professor F. Wood Jones, under the title "The Master Surgeon".

Obituary.

WILLIAM OSCAR DOYLE.

WE regret to announce the death of Dr. William Oscar Doyle, which occurred on February 20, 1935, at Glebe, New South Wales.

Corrigendum.

THE MODERN TREATMENT OF DIABETES MELLITUS.

IN the paper on the modern treatment of *diabetes mellitus* published in the issue of February 23, 1935, two errors requiring correction occurred. On page 232 the word "grammes" in the fourth line should be "milligrammes". On page 236 the fifth paragraph in the second column should read: "If desired, this method can be carried out by a simple modification of the line ration scheme."

XUM

Diary for the Month.

MAR. 1.—Queensland Branch, B.M.A.: Branch.
MAR. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 5.—Tasmanian Branch, B.M.A.: Council.
MAR. 6.—Western Australian Branch, B.M.A.: Council.
MAR. 7.—South Australian Branch, B.M.A.: Council.
MAR. 8.—Queensland Branch, B.M.A.: Council.
MAR. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Ethics Committee.
MAR. 19.—Tasmanian Branch, B.M.A.: Council.
MAR. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 20.—Western Australian Branch, B.M.A.: Branch.
MAR. 22.—Queensland Branch, B.M.A.: Council.
MAR. 26.—New South Wales Branch, B.M.A.: Council, Quarterly.
MAR. 27.—Victorian Branch, B.M.A.: Council.
MAR. 28.—South Australian Branch, B.M.A.: Branch.
MAR. 28.—New South Wales Branch, B.M.A.: Annual.

Books Received.

A HANDBOOK OF GYNÆCOLOGY FOR THE STUDENT AND GENERAL PRACTITIONER, by B. Solomons, B.A., M.D., F.R.C.P.I., F.C.O.G., M.R.I.A.; Third Edition; 1934. London: Baillière, Tindall and Cox. Demy 8vo., pp. 382, with illustrations. Price: 15s. net.

CHRONIC RHEUMATISM, CAUSATION AND TREATMENT, by R. F. Fox, M.D.; F.R.C.P., F.R.Met.S., and J. Van Breemen, D.M.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 371, with eight plates and 38 text figures. Price: 12s. 6d. net.

MANUAL OF CLINICAL LABORATORY METHODS, by P. S. Dimmitt, Ph.G.; 1934. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 165, with illustrations. Price: $2.00 net.

PERIODIC FERTILITY AND STERILITY IN WOMAN: A NATURAL METHOD OF BIRTH CONTROL, by H. Knaus, with a foreword by F. H. A. Marshall, F.R.S.; authorized English translation by D. H. Kitchin and K. Kitchin, M.Sc., M.B., B.S.; 1934. Vienna: Wilhelm Maudrich. Super Royal 8vo., pp. 162, with illustrations. Price: Rm. 15 net.

PHYSICAL DIAGNOSIS, by R. C. Cabot, M.D.; Eleventh Edition; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 564, with illustrations.

PRINCIPLES IN THE TREATMENT OF INFLAMMATION, by T. E. Hammond, F.R.C.S.; 1934. London: H. K. Lewis. Demy 8vo., pp. 220. Price: 10s. 6d. net.

RULES FOR RECOVERY FROM PULMONARY TUBERCULOSIS: A LAYMAN'S HANDBOOK OF TREATMENT, by L. Brown, M.D.; Sixth Edition; 1934. Philadelphia: Lea and Febiger. Crown 8vo., pp. 275. Price: $1.75 net.

SEX AND REVOLUTION, by A. Craig; 1934. London: George Allen and Unwin. Crown 8vo., pp. 144. Price: 4s. 6d. net.

THE ANATOMY OF SURGICAL APPROACHES, by L. C. Kellogg, A.B., M.D.; 1934. London: Baillière, Tindall and Cox. Crown 8vo., pp. 144, with illustrations. Price: 7s. net.

THE RHEUMATIC DISEASES: A CONCISE MANUAL FOR THE PRACTITIONER, by G. D. Kersley, M.A., M.D., M.R.C.P., with a preface by F. R. Fraser, M.A., M.D., F.R.C.P.; 1934. London. William Heinemann (Medical Books) Limited. Demy 8vo., pp. 104, with illustrations. Price: 6s. net.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiii, xiv, xv.

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Medical Officers.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
LOWER BURDEKIN DISTRICT HOSPITAL, AYR, NORTH QUEENSLAND: Resident Medical Officer.
MOOROOPNA HOSPITAL, MOOROOPNA, VICTORIA: Junior Resident Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Pathologist.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Assistant Ophthalmic Surgeon, Relieving Assistant Ophthalmic Surgeon.
TIBOOBURRA DISTRICT HOSPITAL, TIBOOBURRA, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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